Sponsors

The National Asthma Council Australia has received generous support from the following bodies for the publication of this document.

**Government**
Australian Government Department of Health and Ageing

**Corporate**
GlaxoSmithKline Australia, Founding and Principal Sponsor
AstraZeneca
CSL Pharmaceuticals
Merck Sharp and Dohme
Altana Pharma
Novartis
Pharmaxis
Symbion Medical Centres

The *Asthma Management Handbook* has been compiled by the National Asthma Council Australia for use by general practitioners, pharmacists, asthma educators, nurses and other health professionals and healthcare students. The information and treatment protocols contained in the *Asthma Management Handbook* are based on current evidence and medical knowledge and practice as at the date of publication. They are intended as a general guide only and are not intended to avoid the necessity for the individual examination and assessment of appropriate courses of treatment on a case-by-case basis. The National Asthma Council Australia and its employees accept no responsibility for the contents of the *Asthma Management Handbook* or for any consequences of treating asthma according to the guidelines therein.

Published by NATIONAL ASTHMA COUNCIL AUSTRALIA LTD.
ACN 058 044 634
1 Palmerston Crescent, South Melbourne 3205
Tel: 1800 032 495   Email: nac@nationalasthma.org.au
Fax: (03) 8699 0400   Website: www.nationalasthma.org.au

This publication is the sixth handbook on asthma management for medical practitioners distributed by the National Asthma Council Australia. Previous publications were:

*Asthma Management Plan*, 1990, National Asthma Campaign
*Asthma Management Handbook* 1993, National Asthma Campaign
*Asthma Management Handbook* 1996, National Asthma Campaign
*Asthma Management Handbook* 1998, National Asthma Campaign
*Asthma Management Handbook* 2002, National Asthma Council

A handbook for pharmacists, the *Pharmacists’ Asthma Management Handbook*, was also published in 1994.


Any use of the copyright in the National Asthma Council Australia material (hardcopy and electronic versions) must be agreed to and approved by the National Asthma Council Australia and the National Asthma Council Australia must be acknowledged. Such use by commercial organisations will normally attract a fee. However, use of National Asthma Council Australia materials for patient counselling and for educational purposes by not-for-profit organisations will generally be free of charge.


ISSN 1325-4405

The National Asthma Council Australia is the peak body for asthma in Australia. It is a non-profit organisation whose members are The Thoracic Society of Australia and New Zealand, The Royal Australian College of General Practitioners, the Pharmaceutical Society of Australia, Asthma Foundations of Australia and the Australasian Society of Clinical Immunology and Allergy.
Contributors

Guidelines Committee

Professor Justin Beilby (Chairman), general practitioner, SA
Ms Anna Berkelmans, asthma educator, VIC
Dr Chris Brown, general practitioner, QLD
Associate Professor Jo Douglass, allergist and immunologist, VIC
Associate Professor Susan Prescott, paediatric allergist and immunologist, WA
Professor Colin Robertson, paediatric respiratory physician, VIC
Mr Marcus Weidinger, pharmacist, WA
Professor John Wilson, respiratory physician, VIC

Working Group members

Professor Michael Abramson, respiratory physician, VIC
Dr Robert Adams, respiratory physician, SA
Dr Sandra Anderson, respiratory scientist, NSW
Professor Peter van Asperen, paediatric respiratory physician, NSW
Associate Professor Amanda Barnard, general practitioner, ACT
Dr David Batt, general practitioner, SA
Professor Richard Beasley, respiratory physician, NZ
Professor Justin Beilby, general practitioner, SA
Ms Anna Berkelmans, asthma educator, VIC
Dr Simon Bowler, respiratory physician, QLD
Dr Chris Brown, general practitioner, QLD
Dr Shane Brun, general practitioner /sports physician, QLD
Dr Jonathan Burdon, respiratory physician, VIC
Associate Professor Ian Charlton, general practitioner, NSW
Associate Professor Jo Douglass, respiratory physician, allergist and immunologist, VIC
Dr H. John Fardy, general practitioner, NSW
Professor Peter Frith, respiratory physician, SA
Professor Nicholas Glasgow, general practitioner, ACT
Dr Kerry Hancock, general practitioner, SA
Dr Chris Hogan, general practitioner, VIC
Ms Linda Hodge, dietitian, VIC
Professor Alan Isles, paediatric respiratory physician, QLD
Dr Hubertus Jersmann, respiratory physician, SA
Professor Andrew Kemp, allergist and immunologist, NSW
Dr Vicki Kotsirilos, general practitioner, VIC
Dr Karen Latimer, respiratory physician, SA
Dr Doug McEvoy, respiratory physician, SA
Dr Alastair Meyer, emergency physician, VIC
Professor Charles Mitchell, respiratory physician, QLD
Dr Raymond Mullins, allergist and immunologist, ACT
It is a pleasure to present the sixth edition of the *Asthma Management Handbook* to health care practitioners. Like previous editions, the *Asthma Management Handbook 2006* aims to help clinicians and other health professionals make changes in their practice based on sound evidence, and where evidence is lacking, the consensus opinion of Australian experts has been incorporated. The Handbook acknowledges the difficulties of providing organised care in the primary care setting and tries to provide practical strategies that will assist with diagnosis, ongoing management and patient education.

While primarily aimed at GPs, the Handbook is also intended as a resource and teaching tool for community pharmacists, nurses, asthma educators, ambulance officers, consumer representatives and healthcare students.

This edition is the outcome of a long development process begun in January 2004, when the National Asthma Council Australia (NAC) surveyed GPs about their preferences for the next version of the Handbook. The results of that national survey confirmed that GPs still wanted a hard copy publication as well as a web version, and that they sought more information on issues such as allergy in asthma, comorbidities, combination therapies, and systematic care. In June 2004 the NAC convened a multidisciplinary steering committee of experts drawn from health professional bodies: The Thoracic Society of Australia and New Zealand, the Royal Australian College of General Practitioners, the Australasian Society of Clinical Immunology and Allergy, the Pharmaceutical Society of Australia, and from the Australian Asthma and Health Educators’ Alliance.

The Guidelines Committee and other invited opinion leaders resolved that the new *Asthma Management Handbook* should not duplicate existing international guidelines, but build on completed work and provide an Australian context and focus. The group decided to use the GINA, British Thoracic Society (BTS/SIGN) and New Zealand asthma guidelines as a basis, along with a comprehensive literature search to provide subsequent evidence (from 2002 onwards). The search questions were developed to try to provide answers to previously unanswered questions of concern to GPs. This search concentrated on reviews and meta-analyses published in the Cochrane Database and major respiratory journals. The *Asthma Management Handbook 2006* uses the NHMRC levels of evidence (I-IV), which are familiar to most Australian practitioners, supplemented by the tick symbol for practice points based on best practice consensus.

The Guidelines Committee oversaw the work of small ‘chapter working groups’, each with at least two specialists and two GPs to maintain the focus on primary care and practicality. A modified SIGN process was used: working group leaders assessed the evidence and drafted the text, which was then reviewed by other working group members. Additional experts were invited to contribute in particular areas. In all, the Handbook writing and review team comprised over 60 contributors, assisted by two medical writers.

All chapters of the Handbook were internally peer-reviewed among the large group of contributors. The complete draft was then circulated to asthma stakeholder groups, including the NAC’s member bodies, other professional organisations, and the pharmaceutical industry. All comments received were reviewed by the chapter working groups, and by the Guidelines Committee where required, before final amendments were made to the text. The *Asthma Management Handbook 2006* has been endorsed by The Thoracic Society of Australia and New Zealand, the Australasian College for Emergency Medicine and the Royal Australian College of General Practitioners.

---

**Professor Justin Beilby**  
*Chairman*  
*Guidelines Committee*
Managing exacerbations

Acute asthma

Managing acute asthma in adults

Initial assessment

History

Management

Other investigations

Follow-up care after an acute asthma episode

Managing acute asthma in children

Initial assessment

Management

Community-based first aid

Follow-up care after an acute asthma episode

Managing exacerbations

Distinguish exacerbations from poor asthma control

The role of PEF monitoring in detecting exacerbations

Managing exacerbations in adults

Oral corticosteroids

Short-acting beta₂ agonists

Inhaled corticosteroids

Managing exacerbations in children

Oral corticosteroids

Short-acting beta₂ agonists

Inhaled corticosteroids

Leukotriene receptor antagonists

Complementary and alternative medicine in asthma

Clinical evaluation and regulation of CAM

Breathing techniques

Dietary modification

Manual therapies

Acupuncture

Exercise therapies

Medicinal therapies

Psychological therapies

Other therapies

Alternative diagnostic tests

Information resources

Diet and asthma

Food allergy

Food as a trigger for asthma
The role of the practice nurse ................................................................. 79
The role of the community pharmacist .................................................. 80
Demographic considerations: organising your practice to suit your patients ......................................................... 81

**Smoking and asthma** ........................................................................... 82
  Smoking rates in Australia ..................................................................... 82
  Effects of smoking on asthma ............................................................... 83
  Make the car and home a smoke-free zone ......................................... 83
  Mechanisms for effects of smoking on asthma ..................................... 83
  Clinical interventions to help patients quit smoking ......................... 84
  Public policy ....................................................................................... 86

**Chronic obstructive pulmonary disease (COPD) and asthma** ............ 88
  COPD and asthma .............................................................................. 89
  Prevalence ......................................................................................... 89
  Risk factors ....................................................................................... 89
  Diagnosis .......................................................................................... 90
  Management of COPD ....................................................................... 91

**Exercise-induced asthma** ................................................................. 93
  Exercise-induced asthma and exercise-induced bronchospasm .......... 93
  Pathogenesis .................................................................................... 93
  Impact on quality of life, asthma and sporting performance ............... 94
  Detection ......................................................................................... 94
  Assessment of lung function .............................................................. 94
  Under- and over-diagnosis ................................................................. 95
  Effect of training ............................................................................... 95
  Treatment strategies to manage exercise-induced asthma .................. 95
  Drug-free strategies ......................................................................... 96
  Use of asthma medications in competitive sport ................................. 97

**Occupational asthma** ...................................................................... 98
  Definition and mechanism ................................................................. 98
  Incidence, risk factors and prevention ............................................... 99
  Diagnosis ........................................................................................ 99
  Prognosis ....................................................................................... 100
  Management of occupational asthma ............................................... 100
  Further information ...................................................................... 100

**Pregnancy and asthma** ................................................................... 101
  Before pregnancy ............................................................................... 101
  Antenatal care ................................................................................ 102
  Asthma exacerbations during pregnancy ........................................... 102
  Delivery .......................................................................................... 102
  Post-partum phase .......................................................................... 102
  Medications during pregnancy and lactation ..................................... 103
  Smoking ....................................................................................... 103

**Asthma in the elderly** ....................................................................... 104
  Asthma in the elderly is under-diagnosed ......................................... 104
  Diagnosis of asthma in older patients .............................................. 105
  Suggested diagnostic steps in the elderly .......................................... 105
  Identifying patients with airflow limitation ....................................... 105
Excluding diagnoses other than asthma and COPD .................................................................106
Distinguishing asthma from COPD .........................................................................................107
Spirometry in the elderly .........................................................................................................107
The role of a diagnostic treatment trial in the elderly ..............................................................108
Managing asthma in elderly patients .....................................................................................108
Drug treatment .........................................................................................................................108
Patient education .....................................................................................................................109
Review of asthma in the elderly patient .................................................................................109
Acute exacerbations and action plans in elderly patients .......................................................111
Other comorbidities .................................................................................................................112
Obstructive sleep apnoea ........................................................................................................112
OSA in adults ..........................................................................................................................112
OSA in children .......................................................................................................................112
Aetiology of OSA .....................................................................................................................112
Treatment ..................................................................................................................................113
Clinical implications of OSA in asthma management ..............................................................113
Gastro-oesophageal reflux and obesity ..................................................................................113
Gastro-oesophageal reflux ......................................................................................................113
Obesity .....................................................................................................................................114
Asthma and mental illness .......................................................................................................114
Asthma is not a psychosomatic illness ..................................................................................114
Asthma and depression ............................................................................................................114
Asthma and anxiety disorders ..................................................................................................115
Mental illness and adherence to treatment ............................................................................115
Mental illness and smoking .....................................................................................................115
Mental illness and children with asthma ..............................................................................115
Prevention of asthma ..............................................................................................................116
Primary prevention: can the onset of asthma be prevented? ................................................117
Does exposure to environmental tobacco smoke increase asthma risk? .............................117
Does infant feeding affect asthma risk? ................................................................................117
Does allergen avoidance reduce asthma risk? .......................................................................117
Other environmental factors .................................................................................................119
Secondary prevention: can asthma be prevented in patients with other atopic disease? ....120
Children ..................................................................................................................................120
Adults .......................................................................................................................................120
Tertiary prevention: can asthma be cured? ............................................................................120
Appendices
Respiratory function tables ......................................................................................................121
Asthma action plan ..................................................................................................................127
Self-management education checklist ....................................................................................129
First Aid for Asthma .................................................................................................................130
Glossary of asthma terms ........................................................................................................131
Index .......................................................................................................................................134
This sixth edition of the Australian treatment guidelines for asthma, the *Asthma Management Handbook*, gives us a chance to reflect where we have been since The Thoracic Society of Australia and New Zealand first published the Asthma Management Plan in 1989 in the *Medical Journal of Australia*. These were the first national treatment guidelines for asthma published anywhere. The Thoracic Society of Australia and New Zealand asked the newly formed National Asthma Council Australia, the National Asthma Campaign as it then was, to take the guidelines to their main target audience, primary care physicians and other relevant health professional groups. That began an ongoing commitment to the distribution and revision of high-quality, user-friendly principles of asthma care and a thorough implementation process. A survey of medical knowledge acquisition published in the *Medical Journal of Australia* in 1997 found that the asthma treatment guidelines were the second most read reference document after the Antibiotic Guidelines.

Each edition of the *Asthma Management Handbook* has been developed out of extensive consultation especially with the main user group, primary care physicians, but also with respiratory physicians and scientists, allergists, asthma educators, emergency physicians, pharmacists, industry, government and many expert individuals. New developments in asthma care must be evaluated and presented to clinicians in a lucid and unbiased manner. The structure of the sixth edition does not reflect the Six Step Asthma Management Plan but the principles of this are encapsulated in the Ongoing Care chapter. Information is provided on new drug therapies and their changing role, especially combination therapy with an inhaled corticosteroid and a long-acting beta₂ agonist. We have adopted the GINA classifications of asthma severity used by many other countries, and stressed the importance of recognising patterns of asthma. Emphasis is placed on practical interventions that work, including smoking cessation, weight reduction and matching inhaler device with patient capability. Also, attention is drawn to the diagnosis and treatment of co-morbidities including COPD, depression and sleep apnoea, as well as the management of allergic conditions, particularly rhinitis.

For years now, the Handbook has also been available on the National Asthma Council’s much-visited website and its pages are some of the most accessed. From this, the sixth edition, the Handbook will be revised annually on the website.

Over sixty people from around Australia have worked on the Handbook, reviewing evidence, assigning levels of evidence and writing chapters. This is the biggest team effort ever for the NAC on the Handbook, and one cannot speak highly enough of so many dedicated health professionals committed to producing the best possible management guidelines. Much of the evidence assessed has been produced in response to clinical need, by researchers in Australia committed to excellence in clinical care. There is a strong sense of national ownership of the *Asthma Management Handbook*, which is a major contributing factor to the success of its implementation. Government, industry and professional societies have also played an important role in contributing to and reviewing the content.

The National Asthma Council Australia was formed as a coalition many years ago, an excellent and effective model for a national body, and it is this principle of stakeholders working together that has proven to be so effective. A prime example is this *Asthma Management Handbook*. The task of production is indeed enormous and great credit must go to the commitment of the skilled editorial staff of the NAC. We are all proud of this tremendous effort and dedicate the *Asthma Management Handbook 2006* to you all, with many thanks to the Department of Health and Ageing, sponsoring companies and stakeholders.

*Professor John Wilson*
Chairman
National Asthma Council Australia
Levels of evidence and key messages

The Asthma Management Handbook 2006 has been developed by a large multidisciplinary group comprising the NAC Guidelines Committee and expert working groups. Its aim is to provide evidence-based guidance on asthma diagnosis and management. The key recommendations are presented chapter by chapter in a Summary of Practice Points at the beginning of the chapter, along with their NHMRC levels of evidence. The level of evidence highlights the methodology of the studies contributing to the evidence that underpins the guidelines. Where no level I, II, III or IV evidence was available but there was consensus of opinion among the expert group, these clinical practice points have been allocated a tick.

In addition, practical tips based on clinical experience and consensus of expert opinion have been summarised in Practice Tips boxes throughout the text.

References are cited throughout the text. The complete reference lists, chapter by chapter, are available on the Web-based version of the Asthma Management Handbook 2006: www.nationalasthma.org.au

<table>
<thead>
<tr>
<th>Designation of Levels of Evidence – National Health and Medical Research Council*</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
</tr>
<tr>
<td>II</td>
</tr>
<tr>
<td>III-1</td>
</tr>
<tr>
<td>III-2</td>
</tr>
<tr>
<td>III-3</td>
</tr>
<tr>
<td>IV</td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Clinical practice points and practice tips</th>
</tr>
</thead>
<tbody>
<tr>
<td>☑</td>
</tr>
</tbody>
</table>

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
# Asthma in Australia

Over 2.2 million Australians have currently diagnosed asthma.\(^1\)

The prevalence of asthma in Australia is relatively high, by international standards:\(^1\)
- 14-16% of children (one in six)
- 10-12% of adults (one in nine)

Look for signs of allergic rhinitis in patients with suspected asthma.

More boys than girls have asthma. However, after teenage years, asthma is more common in women than in men.\(^1\)

Asthma is more common among Indigenous Australians, particularly adults, than among other Australians.\(^1\)

There is a strong link between asthma and allergy: more than 80% of people with asthma have evidence of allergic sensitisation.\(^2\)

Atopy is strongly associated with asthma that persists beyond the first 6 years of life.\(^3,4\)

The presence of other allergic disorders (eczema or allergic rhinitis) or parental history of atopy are risk factors for persistent asthma at 6 years.\(^5\)

Atopy is also a risk factor for hospitalisation for asthma, as are frequent respiratory symptoms, airway hyperresponsiveness and reduced lung function.\(^6\)

Children aged 0 to 4 years are the group that most commonly visits general practitioners or emergency departments or is hospitalised for asthma.\(^1\)

Among pre-school and primary school-age children, rates of hospital visits for asthma are highest in February.\(^1\)

Around 40% of children who have asthma live with smokers and are likely to be exposed to passive smoke.\(^1\)

Despite the known additional health risks, just as many people with asthma smoke as people without asthma.\(^1\)

People with asthma report poorer general health and quality of life than people without asthma.\(^1\)

More people with asthma suffer from anxiety and depression than people without asthma.\(^1\)

A greater proportion of people with asthma had days away from work or study in the last two weeks (11.4%) than people without asthma (7.9%) preceding a survey.\(^1\)

Poorly controlled asthma restricts participation in normal physical and social activities.\(^7\)

The risk of dying from asthma is highest in the elderly; however, asthma deaths occur in all age groups.\(^1\)

In 2004, 311 people died from asthma – the latest figures.\(^8\)

Asthma deaths are more common among those living in less well-off localities in Australia.\(^1\)

However, education, together with self-monitoring, appropriate drug therapy, regular medical review and a written asthma action plan, reduces morbidity and mortality.\(^7,6\)

Most people with asthma lead normal lives and can participate competitively in sport. Many of Australia’s leading sportsmen and women have asthma.\(^7\)
Definition of asthma

Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, macrophages, neutrophils, and epithelial cells. In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment. The inflammation also causes an associated increase in the existing bronchial hyperresponsiveness to a variety of stimuli.10

Airflow obstruction (excessive airway narrowing) in asthma is the result of contraction of the airway smooth muscle and swelling of the airway wall due to:
• smooth muscle hypertrophy and hyperplasia
• inflammatory cell infiltration
• oedema
• goblet cell and mucous gland hyperplasia
• mucus hypersecretion
• protein deposition including collagen
• epithelial desquamation.

This inflammatory process can cause permanent changes in the airways. Long-term changes include increased smooth muscle, increase in bronchial blood vessels, thickening of collagen layers and loss of normal distensibility of the airway.

Potential triggers for the inflammatory process in asthma include allergy, viral respiratory infections, gastro-oesophageal reflux disease (GORD), irritants such as tobacco smoke, air pollutants and occupational dusts, gases and chemicals, certain drugs, and non-specific stimuli such as cold air exposure and exercise.

Education of people with asthma about the nature of the disease – that it is more than bronchospasm, and is an inflammatory disease – helps them gain a greater understanding of the need for separate types of medication for asthma management:
• bronchodilator (also referred to as reliever) medication
• anti-inflammatory (also referred to as preventer) medication
• long-acting beta; agonist (also known as symptom controller) medication usually prescribed in combination with an inhaled corticosteroid (ICS) preventer.

Combination medications consist of an ICS and a symptom controller in a single inhaler device.

In addition, education about other measures to improve asthma control is important:
• allergen avoidance/control
• use of a written asthma action plan
• smoking cessation, diet and exercise (including specific management of exercise-induced asthma if required).
Diagnosis and classification of asthma in adults

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheeze suggests asthma but is not pathognomonic.</td>
<td>✔</td>
</tr>
<tr>
<td>The absence of physical signs does not exclude a diagnosis of asthma.</td>
<td>✔</td>
</tr>
<tr>
<td>Look for signs of allergic rhinitis in patients with suspected asthma.</td>
<td>✔</td>
</tr>
<tr>
<td>Do not rely on peak flow meters for assessing airflow limitation in the diagnosis of asthma.</td>
<td>✔</td>
</tr>
<tr>
<td>Spirometry is the lung function test of choice for diagnosing asthma and for assessing asthma control in response to treatment.</td>
<td>✔</td>
</tr>
<tr>
<td>Pay close attention to spirometry technique to ensure you get the most reliable readings.</td>
<td>✔</td>
</tr>
<tr>
<td>The absence of acute reversibility of airflow limitation in response to a short-acting bronchodilator does not exclude the diagnosis of asthma.</td>
<td>✔</td>
</tr>
<tr>
<td>Chest X-ray should be ordered if the diagnosis is uncertain, if there are symptoms not explained by asthma, and to exclude other conditions.</td>
<td>✔</td>
</tr>
<tr>
<td>Challenge tests may help confirm a diagnosis of asthma. These should be performed only in specialist facilities.</td>
<td>✔</td>
</tr>
<tr>
<td>Consider allergy testing whenever you diagnose asthma.</td>
<td>✔</td>
</tr>
<tr>
<td>Consider referral to a specialist respiratory physician when the diagnosis is uncertain and for patients in whom occupational asthma is suspected.</td>
<td>✔</td>
</tr>
<tr>
<td>Assess the severity of underlying asthma at the initial visit in a patient with newly diagnosed asthma, then reassess severity classification and/or asthma control at subsequent reviews.</td>
<td>✔</td>
</tr>
</tbody>
</table>

Detection and diagnosis

There is no ‘gold standard’ for the diagnosis of asthma. Recommendations regarding the tools and techniques for asthma diagnosis are based on consensus opinion among respiratory physicians.

The diagnosis of asthma is based on:
- history
- physical examination
- supportive diagnostic testing, including spirometry.

History

The presence of one or more of the following characteristic symptoms is suggestive of asthma:
- wheeze
- chest tightness

- shortness of breath
- cough.

Asthma is especially likely if any of the following applies:
- Symptoms are recurrent or seasonal
- Symptoms are worse at night or in the early morning
- Symptoms are obviously triggered by exercise, irritants, allergies or viral infections
- Symptoms are rapidly relieved by a short-acting bronchodilator (See Diagnostic testing).

However, the symptoms of asthma vary widely from person to person. The absence of typical symptoms does not exclude the diagnosis of asthma.

To detect possible asthma, ask about:
- current symptoms
- pattern of symptoms (e.g. course over day, week or year)
• precipitating or aggravating factors (e.g. exercise, viral infections, ingested substances, allergens)
• relieving factors
• impact on work and lifestyle
• home and work environment
• past history of eczema, hay fever, previous events
• family history of atopy.

In some Aboriginal and Torres Strait Islander communities, asthma is commonly known as ‘short wind’. Health professionals should be aware of this term so as to avoid potential misdiagnosis or confusion with other causes of exertional dyspnoea.

For more information about taking a thorough asthma history, including questions to ask regularly at ongoing review, see Ongoing care.

Examination

Examine the chest for hyperinflation and wheeze. Also look for signs of allergic rhinitis, which commonly co-occurs with asthma, because its presence will affect management. (See Asthma and allergy) Note that:
• wheeze is suggestive, but not diagnostic of asthma
• the absence of physical signs does not exclude a diagnosis of asthma.
• crackles on chest auscultation indicate an alternate or concurrent diagnosis.

Diagnostic testing

Spirometry

Spirometry helps you to diagnose asthma and assess asthma control, by allowing you to:
• assess change in airflow limitation
• measure the degree of airflow limitation compared with predicted normal airflow (or with personal best in patients who have previously undergone spirometry).

Spirometry is the lung function test of choice for diagnosing asthma and for assessing asthma control in response to treatment.
• Single or office-based measurements of peak expiratory flow (PEF) with conventional peak flow meters have significant limitations for assessing airflow limitation.
• A spirometer allows you to verify that the patient has performed the manoeuvre correctly and to generate a precise permanent record of results.
• Most adults, and children over 7 years old, can perform spirometry.

Accurate measurement of respiratory function is necessary to assess and manage asthma. Measurements taken both before and after administration of a short-acting beta₂ agonist (SABA) bronchodilator allow you to:
• diagnose airflow limitation
• measure the degree of airflow limitation
• monitor the effects of treatment
• demonstrate the presence and reversibility of airflow limitation to the patient
• provide objective feedback to the patient about the presence and severity of asthma.

Practice tip

The National Asthma Council Australia recommends that all doctors managing asthma should have access to and use a spirometer.

How to perform spirometry

Clearly explain and physically demonstrate correct spirometry technique:
• Sit upright with feet firmly on the floor and do not lean forward. (Standing is permissible in adults if they find it easier, but not in children.)
• Breathe in until lungs feel absolutely full. (Coaching is essential to do this properly.)
• Form a good seal around the mouthpiece.
• Blast air out as hard and fast as possible and for as long as possible, until the lungs are completely empty.

Repeat the test until you obtain three reproducible and acceptable measures, i.e. all the following apply:
• Forced vital capacity (FVC) of the two highest readings does not vary by more than 150 mL.
• The manoeuvre must be performed with a good start.
• Forced expiration must be maintained throughout the test.
• The patient did not cough during the first second of
the test.

Record the best forced expiratory volume in one second
(FEV1) and FVC obtained. For most people, it is not
practical to make more than eight attempts.

Repeat spirometry at least 10 minutes after giving four
separate doses of salbutamol 100 mg via a metered-dose
inhaler (MDI) and large-volume spacer. (A large-volume
spacer is more efficient.)

Interpreting spirometry

Airflow limitation is judged to be reversible if either of the
following applies:
• Baseline FEV1 >1.7 L and post-bronchodilator FEV1 at
  least 12% higher than baseline
• Baseline FEV1 ≤1.7 L and post-bronchodilator FEV1 at
  least 200 mL higher than baseline.

A similar rule is used to determine reversibility based on
pre-and post-bronchodilator FVC.

Results should be expressed as absolute values and also
as a percentage of predicted values, based on the
patient’s age, height and sex. (See Respiratory function
tables)

Peak expiratory flow measurement

When diagnosing asthma, PEF is not a substitute for
spirometry. (It is useful in the diagnosis of occupational
asthma where very frequent testing is required, and is it
useful way to monitor asthma control for some people.)

Single PEF measurements are not adequate for use in
routine asthma management by doctors.

A peak flow meter is used to detect and measure a
person’s variation in best PEF, in order to assess
variability of airflow limitation. Measurement of PEF:
• is effort-dependent
• varies considerably between instruments.

Isolated readings taken in the surgery or pharmacy with a
meter other than the person’s own must be interpreted
with caution because there is a wide normal range.

Despite its limitations, monitoring of PEF at home or work
is useful when:
• symptoms are intermittent
• symptoms are related to occupational triggers (See
  Occupational asthma)
• the diagnosis is uncertain
• when monitoring treatment response. (See Ongoing
care)

In the absence of an acute bronchodilator FEV1 response,
monitoring of PEF over several days to weeks may be
useful in making a diagnosis.

Diagnosis of asthma

A diagnosis of asthma can be made with confidence in an
adult when:
• the person has variable symptoms (especially cough,
  chest tightness, wheeze and shortness of breath) and
• spirometry shows significantly reversible airflow
  limitation as defined above. (See Interpreting
  spirometry)

Serial PEF measurement over time may also aid in the
diagnosis of asthma. The diagnosis of asthma is
supported if:
• PEF varies by at least 20% (or >60 L/minute) for 3
days in a week over several weeks or
• PEF increases by at least 20% in response to asthma
treatment.

The absence of reversible airflow limitation does not
exclude the diagnosis of asthma. The use of long-acting
beta2 agonists (LABAs) or severe inflammation of the
airways can prevent a response to the SABA
bronchodilator. Repeated measurements, perhaps combined with home measurement of peak expiratory flow, are sometimes necessary to document the presence of asthma.

Other tests

**Practice points**

- Chest X-ray should be ordered if the diagnosis is uncertain, if there are symptoms not explained by asthma, and to exclude other conditions.
- Challenge tests may help confirm a diagnosis of asthma. These should be performed only in specialist facilities.
- Consider allergy testing whenever you diagnose asthma.
- Consider referral to a specialist respiratory physician when the diagnosis is uncertain and for patients in whom occupational asthma is suspected.

**Chest X-ray**

A chest X-ray is not routinely required. An X-ray should be ordered if:
- the diagnosis is uncertain
- there are symptoms not explained by asthma
- there is a need to exclude other conditions such as pneumonia.

**Challenge tests**

Challenge tests may help confirm a diagnosis of asthma and should be performed only in specialist facilities. Referral for appropriate challenge testing may be useful when:
- occupational asthma is suspected
- the diagnosis is uncertain
- the person has not benefited from asthma treatment.

Available challenge tests include the following:
- hypertonic saline
- methacholine
- histamine
- mannitol
- dry air.

**Allergy testing**

Allergy is an important causative factor in asthma and allergy tests should be considered when the diagnosis of asthma is made. For more information, see Allergy and asthma.

**Future tests**

It is likely that tests for sputum eosinophilia and exhaled nitric oxide will become standard components of investigations for diagnosing and monitoring asthma in future. At present these tests are research tools.

**Classification of asthma in adults**

**Practice point**

Assess the severity of underlying asthma at the initial visit in a patient with newly diagnosed asthma, then reassess severity classification and/or asthma control at subsequent reviews.

Assessment of asthma pattern and severity is essential in patients with newly diagnosed asthma to guide initial doses of medications and the frequency of subsequent medical review. Medical review is required more frequently in patients with more severe asthma. Later, the patient’s asthma action plan will be based on ongoing reassessment of asthma classification. See Asthma action plans.

In a patient with newly diagnosed, untreated asthma, asthma is classified (Table 1) according to:
- the frequency of symptoms
- spirometry and post-bronchodilator response.

**Intermittent asthma**

Untreated asthma is classified as intermittent if all the following apply:
- Daytime asthma symptoms occur less than once per week.
- Night-time asthma symptoms occur less than twice per month.
- Exacerbations are infrequent and brief.
- FEV1 is at least 80% predicted and varies by less than 20%.

**Mild persistent asthma**

Untreated asthma is classified as mild persistent if one or more of the following applies (and more severe signs and symptoms are not present):
- Daytime asthma symptoms occur more than once per week but not every day.
- Night-time asthma symptoms occur more than twice per month, but not every week.
• Exacerbations occur occasionally and may affect activity or sleep.
• FEV\textsubscript{1} is at least 80% predicted and varies by 20–30%.

**Moderate persistent asthma**

Untreated asthma is classified as moderate persistent if one or more of the following applies (and more severe signs and symptoms are not present):
• Daytime asthma symptoms occur every day, but do not generally restrict physical activity.
• Night-time asthma symptoms occur at least once per week.
• Exacerbations occur occasionally and may affect activity or sleep.
• FEV\textsubscript{1} is 60–80% predicted and varies by more than 30%.

**Severe persistent asthma**

Untreated asthma is classified as severe persistent if one or more of the following applies:
• Daytime asthma symptoms occur every day and restrict physical activity.
• Night-time asthma symptoms occur every day.
• Exacerbations are frequent.
• FEV\textsubscript{1} is 60% predicted or less, and varies by more than 30%.

The initial assessment must be reviewed once treatment has commenced (Table 2). Ongoing reassessment is based on:
• the amount and type of medication the patient is taking when the symptom profile is observed
• spirometry status at review.

For more information on the ongoing assessment of asthma once the diagnosis has been made, see Ongoing care.

**Note:** “Asthma severity” here refers to the underlying, ongoing status of the stable asthma condition, as distinct from the severity of asthma exacerbations.
• A patient with mild persistent asthma might experience exacerbations ranging in severity from mild to severe. Severe exacerbations in a patient with mild persistent asthma usually occur in the context of multiple triggers, e.g. viral infections and exposure to airborne allergens.
• Asthma classification is a subjective assessment, so there is potential for variation of opinion between doctors. Patients’ perceptions of their asthma severity may also differ from those of their health care providers.

---

**Table 1. Classification of asthma in a patient with untreated, newly diagnosed asthma**

<table>
<thead>
<tr>
<th>Daytime asthma symptoms</th>
<th>Night-time asthma symptoms</th>
<th>Exacerbations</th>
<th>Spirometry</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intermittent</strong></td>
<td>Less than weekly</td>
<td>Less than 2 per month</td>
<td>Infrequent • Brief</td>
</tr>
<tr>
<td><strong>Mild persistent</strong></td>
<td>More than weekly and less than daily</td>
<td>More than 2 per month but not weekly</td>
<td>Occasional • May affect activity or sleep</td>
</tr>
<tr>
<td><strong>Moderate persistent</strong></td>
<td>Daily</td>
<td>Weekly or more often</td>
<td>Occasional • May affect activity or sleep</td>
</tr>
<tr>
<td><strong>Severe persistent</strong></td>
<td>• Daily • Physical activity is restricted</td>
<td>Frequent</td>
<td>Frequent</td>
</tr>
</tbody>
</table>

*Adapted from GINA 2004*

An individual’s asthma pattern (intermittent, mild persistent, moderate persistent or severe persistent) is determined by the level in the table that corresponds to the most severe feature present. Other features associated with that pattern need not be present.
Table 2. **Classification of asthma severity in a patient with treated asthma**

<table>
<thead>
<tr>
<th>Clinical features and lung function</th>
<th>No inhaled ICS</th>
<th>Low-dose ICS</th>
<th>Low- to medium-dose ICS and LABA</th>
<th>High-dose ICS + LABA + other agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any of:</td>
<td>Intermittent</td>
<td>Mild persistent</td>
<td>Moderate persistent</td>
<td>Severe persistent</td>
</tr>
<tr>
<td>• Daytime symptoms occur less than once per week</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Night-time symptoms occur less than twice per month</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Exacerbations are brief</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• FEV₁ between episodes is at least 80% predicted and 90% personal best.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any of:</td>
<td>Mild persistent</td>
<td>Moderate persistent</td>
<td>Severe persistent</td>
<td>Severe persistent</td>
</tr>
<tr>
<td>• Daytime symptoms more than once per week but not every day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Night-time symptoms more than twice per month but not weekly</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• FEV₁ between episodes is more than 80% predicted and 90% personal best.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any of:</td>
<td>Moderate persistent</td>
<td>Moderate persistent</td>
<td>Severe persistent</td>
<td>Severe persistent</td>
</tr>
<tr>
<td>• Daytime symptoms daily</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Night-time symptoms at least weekly</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Exacerbations affect sleep/activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• SABA use daily</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• FEV₁ is 60–80% predicted and 70–90% personal best.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any of:</td>
<td>Severe persistent</td>
<td>Severe persistent</td>
<td>Severe persistent</td>
<td>Severe persistent</td>
</tr>
<tr>
<td>• Daytime symptoms every day and restrict physical activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Night-time symptoms frequent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Exacerbations are frequent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• FEV₁ is less than 60% predicted and less than 70% personal best.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3. **ICS dose equivalents: What is meant by low, medium and high daily doses?**

<table>
<thead>
<tr>
<th>Dose level</th>
<th>CIC*</th>
<th>BDP–HFA**</th>
<th>FP**</th>
<th>BUD**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>80–160 mcg</td>
<td>100–200 mcg</td>
<td>100–200 mcg</td>
<td>200–400 mcg</td>
</tr>
<tr>
<td>Medium</td>
<td>160–320 mcg</td>
<td>200–400 mcg</td>
<td>200–400 mcg</td>
<td>400–800 mcg</td>
</tr>
<tr>
<td>High</td>
<td>320 mcg and above</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 800 mcg</td>
</tr>
</tbody>
</table>

ICS: inhaled corticosteroid; LABA: long-acting beta, agonist; CIC: ciclesonide; BDP–HFA: beclomethasone dipropionate; FP: fluticasone propionate; BUD: budesonide

*ex actuator dose

**ex valve dose

Increase treatment and reassess severity within 3 months

If patient’s asthma has matched this category for 3 months and is stable, consider down-titration of medications and reassess within 3 months.

(For information on adjusting the medication regimen, see Principles of drug therapy)

Asthma severity in a patient with treated asthma is classified according to medications, symptoms and spirometry.
Diagnosis and classification of asthma in children

Asthma in children differs from asthma in adults in clinically important aspects, which include the patterns of asthma, natural history and anatomical factors. The pattern and severity of asthma in childhood vary widely. For the majority of children, asthma will resolve – or at least improve – with age.

Diagnosis

Practice points

- In young children, the diagnosis of asthma can be confirmed by a clinical response to an inhaled bronchodilator.
- In children aged 7 years and over, use spirometry to confirm the diagnosis of asthma.

Asthma in children differs from asthma in adults in clinically important aspects, which include the patterns of asthma, natural history and anatomical factors. The pattern and severity of asthma in childhood vary widely. For the majority of children, asthma will resolve – or at least improve – with age.

Tests to confirm the diagnosis

In young children, the diagnosis of asthma is usually confirmed by a clinical response to an inhaled bronchodilator.

- Only children over 7 years old are likely to be able to perform spirometry or peak expiratory flow (PEF) measurement consistently and reliably, to enable an objective assessment of lung function and bronchodilator response.
- Tests of bronchial hyperresponsiveness are rarely used in children.
- Exercise testing can confirm exercise-induced asthma in children.

Table 1. Other causes of wheeze in young children

<table>
<thead>
<tr>
<th>Condition</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient infant wheezing</td>
<td>Onset in infancy</td>
</tr>
<tr>
<td></td>
<td>No associated atopy</td>
</tr>
<tr>
<td></td>
<td>Associated with maternal smoking</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>Recurrent wheeze and failure to thrive</td>
</tr>
<tr>
<td>Inhaled foreign body</td>
<td>Sudden onset</td>
</tr>
<tr>
<td>Milk aspiration/cough during feeds</td>
<td>Especially liquids</td>
</tr>
<tr>
<td></td>
<td>Associated with developmental delay</td>
</tr>
<tr>
<td>Structural abnormality</td>
<td>Onset at birth</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Associated with congenital heart disease</td>
</tr>
<tr>
<td>Suppurative lung disease</td>
<td>Early morning wet/moist cough</td>
</tr>
</tbody>
</table>

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Alternative diagnoses that are commonly confused with asthma

**Practice tip**

In new patients with a previous diagnosis of asthma, confirm that childhood recurrent non-specific cough has not been wrongly interpreted as asthma.

**Recurrent non-specific cough**

The pathophysiology of recurrent cough in the absence of wheeze appears different from that of classic asthma and should not be confused with asthma. Recurrent non-specific cough is a common symptom in children, particularly preschool-aged children. There is usually no associated atopy or family history of asthma. Usually the child develops a cough in association with an upper respiratory tract infection. Typically:

- cough is dry
- coughing occurs in short paroxysms
- coughing is worse in the early hours of the morning and during exercise
- the paroxysm of coughing may be followed by a vomit.
- episodes last for 2–4 weeks
- cough is non-responsive to therapy.

Recurrent non-specific cough usually resolves by age 6 or 7 years and leaves no residual pulmonary pathology.

When cough is due to asthma, it is usually accompanied by some wheeze and episodes of shortness of breath.

- Cough can be the predominant symptom of asthma, but it is extremely rare for cough to be the only symptom.
- The concept of the “cough-variant asthma” syndrome was popularised in the mid-1980s and it has become an all-embracing label for the symptom of recurrent cough. This has resulted in overdiagnosis of asthma and inappropriate therapy.

**Chronic suppurative lung disease**

The symptoms of chronic suppurative lung disease (chronic bronchitis and bronchiectasis) can be mistaken for those of asthma. Typically, the child with chronic suppurative lung disease presents with a history of cough as the major symptom. The cough usually does not disturb the child’s sleep but typically is most prominent as a moist cough on waking in the morning.

**Exercise-induced respiratory symptoms**

Exercise-induced symptoms are commonly reported in children with asthma.

- Many children with asthma who report exercise-induced dyspnoea fail to demonstrate airflow limitation on formal exercise testing.
- Misinterpretation of dyspnoea as asthma potentially leads to overtreatment.
- Exercise-induced laryngeal dysfunction, characterised by inspiratory stridor or acute air hunger, has recently been described in children and adolescents who are competitive athletes. Unlike vocal cord dysfunction, symptoms of laryngeal dysfunction usually only occur during competitive exercise and are difficult to reproduce in the laboratory.

**Practice points**

- In young children, care is needed to exclude non-asthma causes of wheeze.
- When cough is the predominant symptom of suspected asthma, careful assessment is needed to avoid making an incorrect diagnosis of asthma, or instigating inappropriate management.
- Exercise-induced dyspnoea is not always due to asthma, even in children with a confirmed diagnosis of asthma.
- Asthma management in children should be based on a careful assessment of the pattern of asthma.

**Patterns of asthma in children**

The pattern of asthma determines the need for preventive therapy in children.

**Intermittent asthma**

Intermittent (formerly termed ‘episodic’) asthma is classified as infrequent or frequent.

**Infrequent intermittent asthma**

Infrequent intermittent asthma is the most common pattern, and occurs in 70%–75% of children with asthma (Figure 1). In infrequent intermittent asthma:

- Children have isolated episodes of asthma lasting from 1–2 days up to 1–2 weeks
- Episodes are usually triggered by an upper respiratory tract infection or an environmental allergen
- Episodes are usually more than 6–8 weeks apart and children are asymptomatic in the interval periods.
Severity of infrequent intermittent asthma varies widely. Episodes are usually mild, but children with this pattern account for up to 60% of paediatric hospital admissions.

Children with infrequent intermittent asthma require treatment only during episodes. Regular preventive therapy is not recommended. For more information on the management of infrequent intermittent asthma, see Principles of drug therapy.

**Frequent intermittent asthma**
Approximately 20% of children with asthma have frequent intermittent asthma:
- Episodes occur at intervals shorter than 6–8 weeks.
- Children have minimal symptoms (e.g. exercise-induced wheeze) between episodes.

Frequent intermittent asthma is otherwise similar to infrequent intermittent asthma.

Children with frequent intermittent asthma may benefit from regular preventive therapy with leukotriene receptor antagonists, cromoglycate, nedocromil or low-dose (not greater than 200 mcg per day) inhaled corticosteroids. Preventive treatment is commonly required only during the winter months.

**Persistent asthma**
Approximately 5–10% of children with asthma have persistent asthma:
- Children have symptoms on most days, often including:
  - sleep disturbance due to wheeze or cough
  - early morning chest tightness
  - exercise intolerance
  - spontaneous wheeze.
- Acute asthma episodes may also occur, as for intermittent asthma.

Severity ranges from mild (symptoms 4–5 days per week and readily controlled by low-dose preventive therapy) to severe (frequent severe symptoms and abnormal lung function requiring intensive therapy).

**Asthma classification in children**
Classification of childhood asthma is based mainly on the clinical pattern. In children over 7 years old, spirometry and PEF variability are also useful factors to consider.

Childhood asthma is classified as infrequent intermittent, frequent intermittent, mild persistent, moderate persistent or severe persistent. Patients with asthma of any category can experience mild, moderate or severe exacerbations.

Table 2 represents a reasonable approach to classifying asthma in children over age 5 years as a guide to the rational selection of initial therapy. It is intended as a guide only and the suggested categories are not definitive. Regular review of asthma control and response to treatment is essential to reassess the child’s asthma pattern.

In children under 5 years, asthma diagnosis and classification rely mainly on clinical judgement, taking into account symptoms and physical findings.

For information on assessment of acute asthma in children, see Acute asthma.

Figure 1. Frequency of asthma patterns in children

Three out of four children with asthma have infrequent intermittent asthma, while only one in twenty has persistent asthma. (Proportions differ between primary school-aged and preschool-aged children.)

Adapted from Henderson J, et al 2004

---

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
<table>
<thead>
<tr>
<th>Infrequent intermittent</th>
<th>Nil</th>
<th>Night-time symptoms between exacerbations</th>
<th>Brief Mild Occur less than every 4–6 weeks</th>
<th>More than 80% predicted</th>
<th>Less than 20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequent intermittent</td>
<td>Nil</td>
<td>Night-time symptoms between exacerbations</td>
<td>More than 2 per month</td>
<td>At least 80% predicted</td>
<td>Less than 20%</td>
</tr>
<tr>
<td>Mild persistent</td>
<td>More than once per week but not every day</td>
<td>More than twice per month but not every week</td>
<td>May affect activity and sleep</td>
<td>At least 80% predicted</td>
<td>20–30%</td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>Daily</td>
<td>More than once per week</td>
<td>At least twice per week Restricts activity or affects sleep</td>
<td>60–80% predicted</td>
<td>More than 30%</td>
</tr>
<tr>
<td>Severe persistent</td>
<td>Continual</td>
<td>Frequent</td>
<td>Frequent Restricts activity</td>
<td>60% predicted or less</td>
<td>More than 30%</td>
</tr>
</tbody>
</table>

An individual’s asthma pattern (infrequent intermittent, frequent intermittent, mild persistent, moderate persistent or severe persistent) is determined by the level in the table that corresponds to the most severe feature present. Other features associated with that pattern need not be present.

* Predicted values are based on age, sex and height

** Difference between morning and evening values

FEV<sub>1</sub>: Forced expiratory volume in 1 second; PEF: peak expiratory flow.
Principles of drug treatment in adults

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prescribe reliever therapy for all patients with symptomatic asthma:</strong></td>
<td></td>
</tr>
<tr>
<td>• An inhaled short-acting beta&lt;sub&gt;2&lt;/sub&gt; agonist (SABA) is standard reliever therapy and should be carried by all patients ✓ (except those using the budesonide–eformoterol combination (Symbicort) according to the maintenance and reliever regimen)</td>
<td>✓</td>
</tr>
<tr>
<td>• Eformoterol is an effective reliever in patients using the Symbicort maintenance and reliever regimen. (I) These patients ought not require a separate SABA. ✓</td>
<td></td>
</tr>
<tr>
<td><strong>Treatment with a preventer medication is recommended for patients who have asthma symptoms more than three times per week or use SABA more than three times per week.</strong></td>
<td></td>
</tr>
<tr>
<td><strong>In adults in whom preventer medication is indicated, start with a low dose of inhaled corticosteroids (ICS). Once control is achieved, titrate the dose of ICS to the lowest dose at which effective control of asthma is maintained. Those with moderate persistent asthma will need the addition of a long-acting beta&lt;sub&gt;2&lt;/sub&gt; agonist (LABA).</strong></td>
<td></td>
</tr>
<tr>
<td><em><em>On average, little additional improvement in symptoms or lung function is gained by increasing doses above 800 mcg/day budesonide, 320 mcg/day ciclesonide</em> or 500 mcg/day fluticasone propionate/beclomethasone dipropionate–HFA.</em>*</td>
<td></td>
</tr>
<tr>
<td><strong>Early treatment with ICS in people with persistent symptoms and impaired lung function leads to better lung function in the medium term, and may help prevent the development of irreversible airflow limitation, compared with delayed treatment.</strong> ✓</td>
<td></td>
</tr>
<tr>
<td><strong>In adults, initial therapy with ICS is superior to treatment with a leukotriene receptor antagonist (LTRA), cromone or theophylline for improving lung function and reducing symptoms.</strong></td>
<td></td>
</tr>
<tr>
<td><strong>In adults with moderate-to-severe persistent asthma who experience asthma symptoms despite ICS treatment, the addition of a LABA improves symptom control (I), and reduces ICS requirements (II), compared with ICS alone.</strong></td>
<td>II</td>
</tr>
</tbody>
</table>

*Nominal maximal dose pending further investigation to establish ciclesonide dose–response curve for efficacy

**Note on ICS-equivalents:** Consistent with international guideline publications, stated inhaled corticosteroid doses are expressed in beclomethasone dipropionate (BDP–HFA)-equivalents for simplicity and editorial economy, and do not indicate a recommendation of any particular agent within this drug class. To calculate equivalent doses of other formulations, see Table X: ICS dose equivalents (page 18).
When stepping down combination ICS–LABA therapy, consider ceasing LABA treatment when symptoms are controlled by a daily ICS dose of 100 mcg BDP–HFA or equivalent.

Regular treatment with SABA has no benefit over as-needed use.

**Principles of drug treatment in children and adolescents**

Prescribe a SABA as reliever therapy for all children with symptomatic asthma.

In children, start preventer medication with low-dose ICS, montelukast or inhaled cromone.

Most young children have infrequent asthma episodes, which can be managed with bronchodilators as needed and do not require any long-term preventive medications.

In children, ICS doses greater than 250 mcg BDP–HFA or equivalent should be prescribed only on specialist advice.

Once control is achieved, step down the dose of ICS to the lowest dose at which effective control of asthma is maintained.

There is limited evidence for the efficacy of LABAs in children.

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>The two LABAs differ in onset of therapeutic action: eformoterol has a rapid onset (1–3 minutes) while salmeterol has a slower onset (15–20 minutes).</td>
<td>✓</td>
</tr>
<tr>
<td>• The combination of budesonide plus eformoterol (Symbicort) can be used either as maintenance therapy, or maintenance and reliever therapy.</td>
<td>✓</td>
</tr>
<tr>
<td>• The combination of fluticasone plus salmeterol (Seretide) is used only as maintenance therapy.</td>
<td>✓</td>
</tr>
<tr>
<td>As with ICS alone, combination ICS–LABA therapy should be stepped down to the lowest dose that maintains asthma control.</td>
<td>✓</td>
</tr>
<tr>
<td>When stepping down combination ICS–LABA therapy, consider ceasing LABA treatment when symptoms are controlled by a daily ICS dose of 100 mcg BDP–HFA or equivalent.</td>
<td>✓</td>
</tr>
<tr>
<td>Regular treatment with SABA has no benefit over as-needed use.</td>
<td>I</td>
</tr>
</tbody>
</table>

**SUMMARY OF PRACTICE POINTS LEVEL OF EVIDENCE**

- The combination of budesonide plus eformoterol (Symbicort) can be used either as maintenance therapy, or maintenance and reliever therapy.
- The combination of fluticasone plus salmeterol (Seretide) is used only as maintenance therapy.
- As with ICS alone, combination ICS–LABA therapy should be stepped down to the lowest dose that maintains asthma control.
- When stepping down combination ICS–LABA therapy, consider ceasing LABA treatment when symptoms are controlled by a daily ICS dose of 100 mcg BDP–HFA or equivalent.
- Regular treatment with SABA has no benefit over as-needed use.
The aims of drug treatment are to control symptoms, achieve best lung function and maintain best lung function with the lowest effective doses of medication and the fewest possible adverse effects.

In adults and children, the pattern, severity and level of asthma control will determine which regimen is most appropriate to achieve these aims.

Good asthma control is defined as all of the following:
• Minimal symptoms during day and night
• Minimal need for reliever medication
• No exacerbations
• No limitation of physical activity
• Normal lung function (FEV₁ and/or peak expiratory flow (PEF) > 80% predicted or best).

For those with severe asthma, patients and health professionals may need to consider carefully the trade-off between symptom control, safety (especially the prevention of life-threatening asthma episodes) and the adverse effects and risks of medication.

For more information on interaction between asthma severity and asthma control, see Ongoing care.

**Principles of drug treatment in adults**

An important aim of drug therapy is to **achieve best lung function**. Drug therapy should be commenced or amended appropriate to the level of severity and pattern of asthma symptoms (for further details, see Ongoing care section entitled Assess asthma control regularly).

**All patients** with symptomatic asthma should be prescribed an inhaled rapid-acting beta₂ agonist as short-term reliever therapy.

- A short-acting beta₂ agonist (SABA) is recommended for most patients.
- Those taking the budesonide–eformoterol combination (Symbicort) according to the maintenance and reliever regimen may use this combination as reliever and ought not require a separate SABA.

**Management of intermittent asthma**

An inhaled SABA should be prescribed as short-term reliever therapy for all patients with intermittent (symptoms once weekly or less) asthma.¹ There is currently insufficient high quality evidence to say whether there is any benefit of initiating preventer treatment early in adults with mild intermittent asthma.¹

Consistently requiring SABA more than once daily for symptom relief indicates that a patient has poorly controlled asthma. Patients with high usage of SABA should have their asthma management reviewed.³

Ensure patients understand that decreasing symptom relief from the usual SABA dose indicates worsening asthma. If the patient’s usual dose provides relief of symptoms for less than 3–4 hours, patients should follow their asthma action plan.

**Practice points**

- Prescribe reliever therapy for **all** patients with symptomatic asthma:
  - An inhaled short-acting beta₂ agonist (SABA) is standard reliever therapy and should be carried by all patients (except those using the budesonide–eformoterol combination (Symbicort) according to the maintenance and reliever regimen).
  - Eformoterol is an effective reliever in patients using the Symbicort maintenance and reliever regimen. (I)
  - These patients ought not require a separate SABA.

**Management of persistent asthma**

Most adults with asthma will require ongoing and regular daily management with preventer therapy in addition to as-needed SABA therapy. Preventer therapy with ICS, alone or in combination with LABA, is recommended for patients with mild, moderate or severe persistent asthma. A leukotriene receptor antagonist (LTRA) may be considered as an alternative to ICS where there is reason to avoid ICS or according to patient preference. For further information on classification, see Diagnosis and classification of asthma in adults and Diagnosis and classification of asthma in children.

In individuals with persistent symptoms of asthma, treatment with ICS²,³,⁴
- reduces symptoms
- reduces use of rescue medication
- improves lung function
- decreases exacerbations
- reduces hospital admissions.
When to initiate ICS therapy

Treatment with an inhaled corticosteroid (ICS) should be considered for patients with any of the following:1

- exacerbations of asthma in the last two years
- use of SABA reliever three times a week or more
- asthma symptoms three times a week or more
- waking at least one night per week due to asthma symptoms
- impaired lung function.

Patients with mild persistent asthma may also benefit from regular use of ICS.5,6

Treatment with ICS should not be delayed in people with persistent symptoms and impaired lung function. A degree of residual lung function impairment may persist despite optimal therapy if the initiation of ICS therapy is delayed.

ICS starting dose

The appropriate starting dose of ICS depends on asthma classification.

- In adults with mild-to-moderate asthma, a reasonable starting dose will usually be 80–160 mcg ciclesonide (CIC), 100–200 mcg fluticasone propionate (FP) or beclomethasone dipropionate (BDP–HFA), or 200–400 mcg budesonide (BUD).
- The therapeutic effects of ICS will usually be seen within 3–4 weeks of starting.
- For patients with severe persistent asthma, higher ICS doses (1000 mcg BDP–HFA or equivalent per day) may produce a greater improvement in lung function, but do not necessarily improve symptom control or reduce reliever medication use.
- In individuals using continuous oral corticosteroids, doses of 2000 mcg BDP–HFA or equivalent per day may allow lower doses of oral corticosteroid to be used.

Table 1. ICS dose equivalents: what is meant by low, medium and high daily doses?

<table>
<thead>
<tr>
<th>Daily ICS dose</th>
<th>CIC*</th>
<th>BDP–HFA**</th>
<th>FP**</th>
<th>BUD**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>80–160 mcg</td>
<td>100–200 mcg</td>
<td>100–200 mcg</td>
<td>200–400 mcg</td>
</tr>
<tr>
<td>Medium</td>
<td>160–320 mcg</td>
<td>200–400 mcg</td>
<td>200–400 mcg</td>
<td>400–800 mcg</td>
</tr>
<tr>
<td>High</td>
<td>320 mcg and above</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 800 mcg</td>
</tr>
</tbody>
</table>

ICS: inhaled corticosteroid; LABA: long-acting beta2 agonist; CIC: ciclesonide; BDP–HFA: beclomethasone dipropionate; FP: fluticasone propionate; BUD: budesonide

Practice points

- Treatment with a preventer medication is recommended for patients who have asthma symptoms more than three times per week or use SABA more than three times per week. (I)
- In adults in whom preventer medication is indicated, start with a low dose of inhaled ICS. Once control is achieved, titrate the dose of ICS to the lowest dose at which effective control of asthma is maintained. Those with moderate persistent asthma will need the addition of a long-acting beta2 agonist (LABA). (I)
- On average, little additional improvement in symptoms or lung function is gained by increasing doses above 800 mcg/day budesonide, 320 mcg/day ciclesonide* or 500 mcg/day fluticasone propionate/beclomethasone dipropionate–HFA. (I)
- Early treatment with ICS in people with persistent symptoms and impaired lung function leads to better lung function in the medium term, and may help prevent the development of irreversible airflow limitation, compared with delayed treatment. ✔
- In adults, initial therapy with ICS is superior to treatment with a leukotriene receptor antagonist (LTRA), cromone or theophylline for improving lung function and reducing symptoms. (I)

Review regularly

Asthma should be reviewed regularly and frequently until optimal asthma control has been achieved.

- Assessment of asthma control should include measurement of lung function as well as questioning about recent symptoms. The initial review needs to be undertaken within a few days to few weeks, depending on severity of presenting symptoms.
- It is important to check adherence, demonstrate and review correct use of the medication delivery device(s), assess adverse effects and identify trigger factors. For more information on review of asthma, see Ongoing care.

Adjusting maintenance therapy

Once asthma is controlled, it is recommended that treatment doses be stepped down. Although this back-titration is recommended, it is often not implemented – leaving some patients over-treated. Patients should be
maintained at the lowest effective dose of ICS. There is little evidence to determine the most appropriate way to step down treatment.

- Step-down of medications should be considered after effective control has been in place for 6–12 weeks, decreasing the dose by approximately 25–50% each time.\(^1\)
- The precise time interval and the size of the step-down should be made on an individual basis.
- Reduction in the ICS dose should be slow, over several months, as patients deteriorate at different rates. Regular review of patients as treatment is stepped down is important.\(^1\)
- Always check symptom control and lung function before deciding if another back-titration step is indicated.
- The threshold dose below which ICS should not be reduced has not been defined and will differ between individuals. Some patients are very sensitive to low doses, while others may require a higher dose to maintain asthma control.

**Add-on therapy: role of LABAs**

In patients in whom adequate asthma control is not achieved despite low-dose ICS treatment, a LABA (eformoterol or salmeterol) should be the first choice for add-on therapy after ruling out poor adherence and poor inhaler technique as causes (Figure 1).

The addition of a LABA to ICS improves lung function and symptoms and reduces exacerbations to a significantly greater degree than increasing the dose of ICS alone.\(^7,9,10\)

- Adding a LABA to ICS in symptomatic patients may also ultimately permit use of lower doses of ICS.\(^11\)
- In people taking ICS, LABAs are more effective than the regular use of SABAs in controlling symptoms.\(^3\)
- The duration of a trial of add-on therapy will depend on the target outcome; e.g. prevention of nocturnal awakening may require a relatively short trial of treatment (days or weeks), whereas preventing exacerbations of asthma may require a longer trial of therapy (weeks or months).\(^1\)

The two LABAs differ in onset of therapeutic action: eformoterol has a rapid onset (1–3 minutes) while salmeterol has a slower onset (15–20 minutes).

- The combination of budesonide plus eformoterol (Symbicort) can be used either as maintenance therapy, or as maintenance and reliever therapy.
- The combination of fluticasone plus salmeterol (Seretide) is used only as maintenance therapy.

**Adjusting the ICS-plus-LABA regimen**

The principle of maintaining asthma control with the lowest effective dose applies to patients using ICS and LABA in combination, irrespective of whether the two agents are delivered via separate inhalers or via a single fixed-dose inhaler.

- When deciding which drug to step down first and at what rate, the severity of asthma, treatment-related adverse effects, achieved therapeutic benefits and the patient’s preference should all be taken into account.\(^1\)
- When down-titrating ICS—LABA combination therapy, consider ceasing LABA treatment when symptoms are controlled for at least 3 weeks on a daily ICS dose of 100 mcg BDP–HFA or equivalent. (IV)
- Regular treatment with SABA has no benefit over as-needed use. (I)

**Fixed-dose ICS–LABA combination therapy regimens.**

- The combination of fluticasone plus salmeterol (Seretide) is used only as maintenance therapy.

**Practice points**

- In adults with moderate-to-severe persistent asthma who experience asthma symptoms despite ICS treatment, the addition of a LABA improves symptom control (I), and reduces ICS requirements (II), compared with ICS alone.
- The two LABAs differ in onset of therapeutic action: eformoterol has a rapid onset (1–3 minutes) while salmeterol has a slower onset (15–20 minutes).
- The combination of budesonide plus eformoterol (Symbicort) can be used either as maintenance therapy, or as maintenance and reliever therapy. ✓
- The combination of fluticasone plus salmeterol (Seretide) is used only as maintenance therapy. ✓
- As with ICS alone, combination ICS–LABA therapy should be stepped down to the lowest dose that maintains asthma control. ✓
- When stepping down combination ICS–LABA therapy, consider ceasing LABA treatment when symptoms are controlled by a daily ICS dose of 100 mcg BDP–HFA or equivalent. (IV)
- Regular treatment with SABA has no benefit over as-needed use. (I)
Figure 1. **Management of mild, moderate and severe persistent asthma**

- **Mild persistent asthma**
  - ICS alone (200 mcg BDP–HFA or equivalent daily)*

- **Moderate persistent asthma**
  - Add LABA to ICS

- **Severe persistent asthma or Persistent symptoms or poor lung function on ICS alone (200 mcg BDP–HFA daily or equivalent)**
  - Increase ICS dose to 400–500 mcg BDP–HFA daily or equivalent
  - Consider ICS dose reduction by 25–50 %

- **Well-controlled asthma**
  - Consider ceasing LABA

---

* LTRA can be used as an alternative to low-dose inhaled corticosteroids when a non-corticosteroid therapy is preferred.  

Adapted from Powell H, Gibson PG, 2003b12 and Douglass JA, Reddel HK, 200513  
The steps are made at intervals of 6–12 weeks. Regimens listed are in addition to as-needed reliever medication  

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
If the response to add-on therapy is inadequate

- If there is some response to LABA but control remains poor, increase the dose of ICS and continue with the LABA.¹
- If there is no response to LABA, reconsider the diagnosis.
- If control is still inadequate after a trial of LABA and after increasing the dose of inhaled steroid, consider a sequential trial of add-on therapy (i.e. LTRA, theophylline, slow-release beta2 agonist tablets).

Effect of LABA use on SABA efficacy

Regular use of LABAs can reduce the bronchodilatory response to SABAs,¹⁴⁻²⁰ but the clinical importance of this effect is unclear. Careful review of asthma control is necessary in each patient to assess whether SABA is effective. Ongoing surveillance is needed to determine whether LABA-induced sub-sensitivity to SABA is clinically significant at the population level. Based on current evidence, there is no reason to expect LABA use to increase the risk of life-threatening asthma episodes in Australia, where LABA monotherapy is not recommended in evidence-based guidelines.

Until further evidence is available, patients using a LABA:
- may sometimes need to use more actuations of SABA to relieve bronchoconstriction than patients not using a LABA
- should be advised to use enough SABA to relieve symptoms, and told that they may need more reliever medication than before they started the LABA
- should be reminded to have a reliever inhaler available, and to use it if their asthma symptoms worsen.

Fixed-dose ICS–LABA combination therapy regimens

Combination therapy products are available that deliver both ICS and LABA in a single inhaler. See Drugs and devices for specific product details. There are no clinical differences in efficacy or adherence between delivery of ICS and LABA in a combination inhaler and delivery in separate inhalers.¹⁰ However, single-inhaler therapy may be more convenient and cheaper for patients.

General principles using a fixed-dose ICS–LABA combination inhaler

Currently, the strongest evidence of a benefit with fixed-dose combination inhaler is in patients who are already taking daily ICS and are symptomatic on moderate doses of ICS (200–400 mcg BDP–HFA or equivalent). However, significant effects may also be seen in patients taking lower doses of ICS.¹⁰

The method of titrating the dose of fixed-dose combination treatment and adjusting treatment in response to worsening asthma control depends on which combination is used. See Dose adjustment with fluticasone plus salmeterol (Seretide) and Dose adjustment with budesonide plus eformoterol (Symbicort).

The following principles apply to the use of both available fixed-dose combination inhalers.

Gaining control

The initial dose of combination therapy used may be higher than the final maintenance dose. The aim is to gradually reduce the dose of combination medication once control is achieved. See Back-titrating single-inhaler combination therapy.

Assessing asthma control

Assess asthma control 6–12 weeks after commencing a combination inhaler.
- If patients experience persistent symptoms or continue to require reliever medication daily, consider other contributing causes/triggers and/or specialist referral.
- Current evidence does not support exceeding recommended doses of ICS or LABA.
- Adherence and inhaler technique should be checked at every visit.

Back-titrating combination therapy

- In patients whose asthma is well controlled and stable on combination therapy, step down the ICS dose to the minimum effective dose. (In those using fluticasone plus salmeterol, this may require the addition of a separate ICS inhaler to facilitate gradual reduction of the ICS component.)
- Consider withdrawal of the LABA if asthma remains well controlled and stable on a fixed-dose combination that includes not more than 100 mcg BDP–HFA (or equivalent) daily. There is no evidence from clinical trials to recommend a specific strategy, but LABA cessation and advice to take SABA as needed
appears appropriate. Following this strategy, if SABA use is three times a week or more, the patient is likely to remain better controlled on combination therapy.

• Maintain at the lowest effective ICS dose and reinforce trigger factor avoidance and management.
• Schedule a follow-up appointment to assess the appropriate dose of each component.

Dose adjustment with fluticasone plus salmeterol (Seretide)
When initiating therapy with fluticasone plus salmeterol fixed-dose inhaler, advise patients to keep their ICS inhaler; it may be required when reducing the dose of combination therapy to a maintenance level, or to manage a deterioration in asthma control.

Dose adjustment with budesonide plus eformoterol (Symbicort)
The budesonide–eformoterol combination can be used according to either a maintenance regimen or maintenance-and-reliever regimen.

Standard maintenance regimen
In patients using the standard maintenance regimen, day-to-day symptoms are managed using as-needed SABA.
In response to worsening asthma control, the dose of the maintenance regimen can be increased and later stepped down again when control is gained. See General principles using a fixed-dose ICS–LABA combination inhaler.

Maintenance-and-reliever regimen
Fixed-dose budesonide plus eformoterol can be used as both reliever and maintenance medication.
• In response to asthma symptoms, additional doses can be used on an as-needed basis up to 72 mcg eformoterol per day. See Drugs and devices.
• Additional as-needed doses in response to symptoms necessarily result in a higher daily ICS dose.

Other add-on therapy options
When added to ICS treatment:1
• leukotriene receptor antagonists provide small improvements in lung function, decrease in exacerbations and improvement in symptoms.
• theophyllines improve lung function and symptoms, but side-effects are common.
• addition of short-acting anticholinergics is generally of no value.

• addition of cromones as maintenance therapy is of no benefit in most patients.
• slow-release beta, agonist tablets improve lung function and symptoms, but side-effects are common.

Principles of drug treatment in children and adolescents

Practice points
• Prescribe a SABA as reliever therapy for all children with symptomatic asthma.
• In children, start preventer medication with low-dose ICS, montelukast or inhaled cromone. (III)
• Most young children have infrequent asthma episodes, which can be managed with bronchodilators as needed and do not require any long-term preventive medications.
• Most young children have infrequent asthma episodes, which can be managed with bronchodilators as needed and do not require any long-term preventive medications.
• In children, ICS doses greater than 250 mcg BDP–HFA or equivalent should be prescribed only on specialist advice.
• Once control is achieved, step down the dose of ICS to the lowest dose at which effective control of asthma is maintained.
• There is limited evidence for the efficacy of LABAs in children. (I)

Most young children have infrequent asthma episodes, which can be managed with bronchodilators as needed. They do not require any long-term preventive medications.
However, for children of any age who have obvious and recurrent asthma symptoms, anti-inflammatory agents are currently the most effective long-term preventive medications and are effective in reducing asthma attacks (Figure 2).
Whilst the efficacy of ICS in children has been clearly demonstrated, the dose–response curves have not been well described over the range of asthma severities seen in children. The lowest dose of ICS compatible with maintaining disease control should be used.1
• In children with frequent intermittent and mild persistent asthma, use inhaled cromones, oral LTRAs or low-dose ICS.21–23
• For children with moderate-to-severe persistent asthma, an ICS is the preferred option.21
• LABAs can be prescribed in children in combination with ICS (salmeterol in children 5 years or eformoterol in children 12 years and older). However, there is limited evidence for their efficacy and safety in children.22

For information on the management of adolescents with asthma, see Ongoing care.

Stepwise approach to drug therapy in children

Start treatment at the step most appropriate to the level of asthma severity and step up or down as necessary.21
• Step up if control is not achieved or sustained. Improvement should be evident within one month. Review the child’s medication technique, compliance, and avoidance of risk factors before changing the medication dose.
• Step down if control is sustained for at least 3 months; follow a gradual stepwise reduction in treatment.
• The goal is to decrease treatment to the least medication necessary to maintain control.
• Review treatment every 3–6 months once asthma is under control.
• Consider referral to an asthma specialist if other conditions complicate asthma (e.g. sinusitis) or the child does not respond to therapy.

Wheezing

In infants, wheezing is often not due to asthma but rather due to acute viral bronchiolitis or transient early wheeze. Therefore, the response to inhaled bronchodilators is not generally as beneficial as in older children. See Ongoing care.
• Many children aged 2–5 years with wheeze do not have classical asthma. Many have ‘viral-induced wheeze’ associated with a respiratory infection or wheezy bronchitis.
• These children are well in the interval between episodes, are non-atopic and have a good prospect of outgrowing the tendency to wheeze in later childhood.
• The standard approach for these patients is to use bronchodilators as needed. Neither ICS nor oral steroids appear to be helpful, but LTRAs may be useful both as a long-term preventive agent and as an ‘episode modifier’.24
Figure 2. Preventer therapy in children

Montelukast or inhaled cromones

Low-dose ICS
(FP/BDP–HFA 100–200 mcg/day
BUD 200–400 mcg/day)

or

Low-dose ICS
(FP/BDP–HFA 100–200 mcg/day
BUD 200–400 mcg/day)

Check:
Diagnosis
Technique
Adherence
Remember:
Back titration

Increase ICS
(FP/BDP–HFA 200–250 mcg/day
BUD 400–800 mcg/day)

Add LABA

Further increase dose of ICS to maximum
(FP/BDP–HFA 500 mcg/day
BUD 800 mcg/day)
There are three main groups of asthma medications:
- Relievers
- Preventers
- Symptom controllers – usually prescribed in combination with an inhaled corticosteroid (ICS) preventer.

Combination medications consist of an ICS and a symptom controller in a single inhaler device.

**Relievers**

Relievers have a direct bronchodilator effect and relieve the symptoms of asthma. They are the mainstay drugs for the acute relief of asthma symptoms. Relievers are short-acting beta₂ agonists (SABAs) and eformoterol, a long-acting beta₂ agonist (LABA) with rapid onset of action.

**Short-acting beta₂ agonists**

- e.g. salbutamol (*Airomir, Asmol, Epaq, Ventolin*);
- terbutaline (*Bricanyl*) (blue inhalers)

Short-acting beta₂ agonists (SABAs) such as salbutamol and terbutaline relax bronchial smooth muscle by stimulating beta₂-receptors, primarily in the airways, skeletal muscle and, to a lesser extent, the heart (this is especially important at higher doses). This accounts for adverse effects such as tachycardia and tremor.

SABAs treat the immediate symptoms of asthma, and can help to prevent exercise-induced asthma when used before exercise. They have no anti-inflammatory properties. They should be taken on an as-needed or on-demand basis, rather than regularly. Salbutamol and terbutaline will work within 5–15 minutes of inhalation.

**Indications**

- Acute relief of asthma symptoms
- Symptom relief during maintenance treatment of asthma
- Protection against exercise-induced asthma.

**Delivery**

- Inhalation is the preferred method of delivery via a pressurised metered-dose inhaler (MDI), breath-activated inhaler or dry-powder inhaler (DPI).
- The onset of action is faster with inhalation and there are fewer adverse effects compared to other delivery methods.
- Delivery via an inhaler plus a spacer is as effective as nebulised therapy, with less time to deliver a dose and reduced equipment maintenance. See Acute asthma.
- Use a valved spacer for adults and older children, a spacer with an attached face-mask or mouthpiece for children aged 2–4 years and a large-volume spacer for children aged 5 years and over.
- Drug delivery via spacer is reduced by multiple actuations of the aerosol device. Optimal delivery is obtained by using one actuation at a time. This also allows for the recovery time of the valve mechanism. The static electricity charge on plastic spacers can also reduce delivery. This effect is reduced after initial use of the spacer device or by washing before first use and then at least every month; hand-wash in warm water with kitchen detergent without rinsing and allow to air-dry. Avoid storage in a plastic bag. Some, but not all spacers are dishwasher safe and cleaning should be in accordance with manufacturer’s recommendations in each instance.
- Give the patient specific instructions about the dosage to be used for minor and acute exacerbations.
- Oral therapy with SABAs is discouraged in all age groups due to a slower onset of action and the higher incidence of behavioural side effects and sleep disturbance. It may have a limited role in the treatment of children under 2–3 years of age with mild occasional asthma.

---

**Note on ICS-equivalents:** Consistent with international guideline publications, stated inhaled corticosteroid doses are expressed in beclomethasone dipropionate- (BDP–HFA)-equivalents for simplicity and do not indicate a recommendation of any particular agent within this drug class. To calculate equivalent doses of other formulations, see Table 1. ICS dose equivalents (page 27).
**Short-acting beta₂ agonists**

<table>
<thead>
<tr>
<th>Dosage Form</th>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metered-dose inhaler</td>
<td>Salbutamol</td>
<td>100 mcg/inhalation, 1–2 inhalations as required every 3–6 hrs For acute symptoms: 4–6 inhalations if &lt; 6 yrs; 8–12 inhalations if &gt; 6yrs; if necessary repeat in 20 minutes</td>
</tr>
<tr>
<td>Turbuhaler</td>
<td>Terbutaline</td>
<td>500 mcg/inhalation, 1 inhalation as required every 3–6 hrs</td>
</tr>
<tr>
<td>Autohaler</td>
<td>Salbutamol</td>
<td>100 mcg/inhalation, 1–2 inhalations as required every 3–6 hrs</td>
</tr>
<tr>
<td>Single-dose nebuliser units</td>
<td>Salbutamol</td>
<td>Children 4–12 years: 2.5 mg unit every 3–6 hrs Children &gt; 12 years and adults: 5 mg unit every 3–6 hrs Terbutaline Children 4–12 years: 2.5 mg (1 mL) every 3–6 hrs Children &gt; 12 years and adults: 5 mg (2 mL) every 3–6 hrs</td>
</tr>
<tr>
<td>Nebuliser solutions</td>
<td>Salbutamol 5 mg/mL</td>
<td>Children: 0.03 mL/kg/dose to a maximum of 1 mL, diluted with saline to 4 mL, every 3–6 hrs Adults: 1 mL every 3–6 hrs Terbutaline Children: 0.08 mL/kg/dose every 4–6 hrs Adults: 1–2 mL/dose every 3–6 hrs</td>
</tr>
</tbody>
</table>

**Long-acting beta₂ agonist with rapid onset**

Eformoterol can be used as a reliever within the Symbicort maintenance and reliever regimen only. See Combination medications.

**Ipratropium bromide**

(Apoven 250, Atrvent, DBL Ipratropium, Ipratrin, Chemart Ipratropium, GenRx Ipratropium, Healthsense Ipratropium, Terry White Chemists’ Ipratropium) (green inhalers)

Ipratropium bromide is an inhaled anticholinergic bronchodilator with a slower onset of action (30–60 minutes) than other relievers.

- Its major role is in the treatment of chronic obstructive pulmonary disease (COPD). For more information on the management of COPD see COPD and Asthma.
- Ipratropium bromide has a limited place in the day-to-day management of people with asthma, although in children the addition of ipratropium bromide to a SABA has shown benefit in the initial management of moderate and severe acute asthma.
- Administration by MDI and spacer is preferable to nebuliser therapy wherever possible.
- Adverse effects are primarily oropharyngeal and relate to its anticholinergic effects e.g. dry mouth, throat irritation.
- To reduce potential for ocular anticholinergic effects (dryness, irritation, blurred vision, visual halos), advise patients using the nebuliser solution to wear eye protection.

**Theophylline**

(Austyn, Nuelin)

Theophylline is thought to benefit asthma via bronchial smooth muscle relaxation, anti-inflammatory effects and increased diaphragm contractility. The anti-inflammatory effects occur at lower concentrations than required for bronchodilation.

- The role of theophylline in the treatment of asthma has declined. It should not be used as firstline therapy in asthma.¹

---

¹Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
• It is associated with more frequent adverse effects than other relievers, and is less effective than salmeterol in improving lung function, relieving both night and day symptoms and reducing the need for rescue therapy.
• Theophylline has a narrow therapeutic index and variable metabolism.

Indications
• Treatment of severe acute asthma
• Maintenance treatment in patients with severe persistent asthma who require multiple drugs to achieve symptom control.

Concentration monitoring
Monitor concentration at initiation of treatment, if drug regimen is changed, if adverse effects are suspected or if patient stops or starts smoking (altered drug metabolism). Therapeutic range:
• Trough (pre-dose) theophylline plasma levels are generally accepted to be 10–20 mg/L (55–110 micromol/L)
• Plasma concentrations at the lower end of the range e.g. 9–10 mg/L (50–55 micromol/L) appear to be effective in some patients.

Some proprietary products also contain theophylline (e.g. choline theophyllinate in Brondecon elixir), which may need to be considered.

Preventers
Preventer agents have anti-inflammatory properties and are generally taken regularly to reduce symptoms and exacerbations. These include:
• ICS: beclomethasone dipropionate, budesonide, fluticasone propionate and ciclesonide.
• Leukotriene receptor antagonists (LTRAs): montelukast
• Cromones: cromoglycate and nedocromil.

Oral or parenteral corticosteroid agents are potent anti-inflammatory agents reserved for use in acute or very severe chronic asthma.

Inhaled corticosteroids
e.g. beclomethasone dipropionate—HFA (Qvar) (brown inhaler); budesonide (Pulmicort) (brown inhaler); fluticasone propionate (Flixotide) (orange inhaler); ciclesonide (Alvesco) (rust-coloured inhaler)

Table 1. ICS dose equivalents: what is meant by low, medium and high daily doses?

<table>
<thead>
<tr>
<th>DOSE LEVEL</th>
<th>CIC*</th>
<th>BDP–HFA**</th>
<th>FP**</th>
<th>BUD**</th>
</tr>
</thead>
<tbody>
<tr>
<td>LOW</td>
<td>80–160 mcg</td>
<td>100–200 mcg</td>
<td>100–200 mcg</td>
<td>200–400 mcg</td>
</tr>
<tr>
<td>MEDIUM</td>
<td>160–320 mcg</td>
<td>200–400 mcg</td>
<td>200–400 mcg</td>
<td>400–800 mcg</td>
</tr>
<tr>
<td>HIGH</td>
<td>320 mcg or above</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 800 mcg</td>
</tr>
</tbody>
</table>

ICS: inhaled corticosteroid; LABA: long-acting beta agonist; CIC: ciclesonide; BDP–HFA: beclomethasone dipropionate; FP: fluticasone propionate; BUD: budesonide

*ex actuator dose
**ex valve dose

ICS remain the most effective agents for gaining and maintaining control of asthma in adults and in children with persistent asthma.

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
• The use of ICS has been associated with lower asthma mortality rates and a reduced need for hospitalisation, as well improvement in quality of life for children and adults with asthma.2
• Early treatment with ICS in people with persistent symptoms and impaired lung function leads to better lung function in the medium term, and may help prevent the development of irreversible airflow limitation.
• ICS have a relatively flat dose response curve for efficacy in symptoms and lung function.3,4 At a group level, little additional benefit in symptoms or lung function is gained from doses above 320 mcg/day of CIC, 500 mcg/day of FP/BDP–HFA or 800 mcg/day of BUD.
• There is an increased risk of cataracts, reduced bone mineral density, osteoporosis, glaucoma and bruising of the skin with long-term treatment with high-dose ICS.
• In adults with asthma who have pre-existing conditions such as osteoporosis or cataracts, the need for high doses of ICS should be balanced against the risk of further systemic side effects.
• The daily dose should be titrated according to the patient’s clinical response and lung function. The delivery device may influence the final dose.
• There is no need to routinely use a SABA immediately before taking preventer medication.

Safety of ICS

The risk of adverse effects is dose-related but there is also some individual patient sensitivity to the effects of corticosteroids. It is important to find a balance between benefits and risks for each patient. Consider the patient’s use of other systemic steroids when assessing steroid risk.

Adults

• There is little evidence that doses below 500 mcg per day BDP–HFA or equivalent cause any short-term adverse effects apart from the local side-effects of dysphonia and oral candidiasis.
• A systematic review of ICS found that the number needed to harm (NNH) for development of hoarseness/dysphonia at 200 mcg FP daily was 131, whereas the NNH for a daily dose of 500 mcg FP was 23.4 Similarly, the NNH for oral candidiasis was 61 at 200 mcg FP daily, and 21 with 500 mcg FP daily.
• A systematic review reported no effect on long-term bone density at doses up to 500 mcg BDP–HFA daily or equivalent, although further studies are investigating long-term safety.5
• Osteoporosis screening is advised for adults on long-term high-dose ICS.
• The significance of small biochemical changes in adrenocortical function is unknown.5
• Cataracts may occur in older patients with high cumulative doses of ICS.

Children

• Administration of inhaled corticosteroids at or above 200 mcg of BDP–HFA daily or equivalent may be associated with systemic side-effects e.g. growth failure and adrenal suppression, although isolated growth failure is not a reliable indicator of adrenal suppression.5
• The amount of growth suppression is likely to be a maximum of 1 cm and is non-progressive.
• Poorly controlled asthma can also cause growth suppression.
• Children using regular ICS should have their height monitored on a regular basis.
• Clinical adrenal insufficiency has recently been identified in a small number of children, who have become acutely unwell at the time of intercurrent infectious illness.5 Most of these children were over-treated with ICS often because of over-reliance on cough to diagnose asthma or to assess its severity.
• Consider the possibility of adrenal insufficiency in any child maintained on ICS presenting with shock or a decreased level of consciousness; serum biochemistry and blood glucose levels should be checked urgently. Consider whether parenteral hydrocortisone is required.
• The relative benefits and risks of inhaled corticosteroids in children should be assessed on an individual basis and must be balanced against the risks and morbidity of poorly controlled asthma.

Beclometasone dipropionate–HFA (Qvar)

Beclometasone has a low hepatic first-pass mechanism and an active metabolite, which results in some systemic bioavailability. Qvar is a CFC-free preparation. The finer particle size results in greater intrapulmonary deposition than the CFC preparations, so a lower dose is required.
**Budesonide (Pulmicort)**

Budesonide has been approved for once-daily use in adults with asthma controlled by 400 mcg or less of ICS per day. Its potency is approximately half that of BDP–HFA and FP. Budesonide has Category A listing for pregnancy.

Budesonide is available in a *Turbuhaler* device and a nebulised suspension. Budesonide combined with eformoterol is available as the combination inhaler *Symbicort*. See *Combination medications* for further information.

**DOSAGE**

**Turbuhaler**  
100 mcg, 200 mcg, 400 mcg/inhalation  
Adults: 400–2400 mcg/day  
Children: 200–800 mcg/day  

**Respules**  
For nebulised therapy:  
RESPULES 0.5 mg per 2 mL and 1 mg per 2 mL  
Adults: 0.5–2 mg twice daily  
Children: 0.25–0.5 mg twice daily

**Fluticasone propionate (Flixotide)**

Fluticasone propionate has equivalent potency to BDP–HFA and approximately twice the potency of budesonide when given through comparable devices. It has negligible oral bioavailability since the portion of the dose that is swallowed is subject to extensive first-pass metabolism.

Fluticasone is available as an MDI and a dry powder for inhalation via *Accuhaler*. Fluticasone combined with salmeterol is available in the combination inhaler *Seretide*. See *Combination medications* for further information.

**Ciclesonide (Alvesco)**

Ciclesonide, a relatively new corticosteroid agent, is indicated for the prophylactic management of asthma in adults and in children over 12 years of age. It is delivered as an ultra-fine aerosol, which may facilitate intrapulmonary deposition.

Clinical trials have shown that ciclesonide is well tolerated and effective in treating asthma of varying disease severity in adults and adolescents. Safety and efficacy was maintained over 12 months. Treatment within recommended doses did not cause HPA axis suppression as measured by 24-hour serum and urine cortisol concentrations or cosyntropin tests.

**DOSAGE**

Recommended starting dose of ciclesonide for patients previously maintained on bronchodilator therapy alone: 80 mcg once daily.

Patients previously maintained on another inhaled corticosteroid may require a higher dose depending on their current maintenance dose.

**MDI**  
80 mcg/dose, 160 mcg/dose, 120 doses per MDI  
Adults and children > 12 yrs: 80–320 mcg daily  
Administration 1–2 puffs once daily, morning or evening  
Spacer device not required

**Leukotriene receptor antagonists**

*e.g.* montelukast sodium (*Singulair*)

**Indications**

- Prevention of day and night-time symptoms of asthma
- Treatment of aspirin-sensitive asthma patients
- Prevention of exercise-induced bronchoconstriction
- As add-on therapy to ICS when LABAs are not tolerated, or when inadequate control is achieved.
LTRAs in adults

LTRAs are less effective than low-to-moderate doses of ICS in reducing symptoms such as night-time waking and the need for rescue medication. They are also less effective for improving lung function and quality of life than ICS.7

- The addition of a LTRA to ICS improves symptom control compared to ICS alone, but the improvement is less than that achieved when a LABA is added to an ICS.1,8,9
- LTRAs do not provide immediate benefit for acute episodes of asthma.
- LTRAs may have a preventive role in aspirin and exercise-induced asthma and in those who cannot take inhaled therapy.8,10

LTRAs in children

LTRAs can be used as sole therapy in children with frequent intermittent or mild persistent asthma. LTRAs are equally effective as low-dose ICS in mild persistent asthma.11 LTRAs reduce the frequency of exacerbations in young children.12 LTRAs have a number of potential advantages, particularly in children, including:

- Oral administration
- Once daily administration
- Effective against exercise-induced asthma
- Relatively free of adverse effects
- Likely to benefit both the asthma and associated allergic rhinitis.

Montelukast sodium (Singulair)

Montelukast is indicated for the prophylaxis and treatment of asthma in adults and in children aged 2 years and over.

<table>
<thead>
<tr>
<th>DOSAGE</th>
<th>10 mg, 5 mg, 4 mg (paediatric, chewable)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tablet</td>
<td></td>
</tr>
<tr>
<td>Adults and children</td>
<td>10 mg nocte</td>
</tr>
<tr>
<td>&gt; 15 years:</td>
<td>Children 6–14 years: 5 mg chewable tablet nocte</td>
</tr>
<tr>
<td>Children 2–5 years:</td>
<td>4 mg chewable tablet nocte</td>
</tr>
</tbody>
</table>

Montelukast may be taken with or without food.

Cromones

e.g. sodium cromoglycate (Intal Forte CFC-free) (white inhaler); nedocromil sodium (Tilade CFC-free) (yellow inhaler)

Sodium cromoglycate (Intal Forte CFC-free)

Sodium cromoglycate inhibits the immediate and late response to allergen challenge and may therefore be useful if used before allergen exposure in susceptible individuals.

- It requires more frequent administration than ICS and the onset of benefit may be slow.
- Sodium cromoglycate has few, if any, adverse effects. Therapeutic effect is usually obvious within 1–2 weeks but a 4-week trial is recommended before considering other treatments.

Indications

Sodium cromoglycate may be used as initial preventive therapy for children with frequent episodic to mild persistent asthma, and may be of some benefit in adults with mild asthma.

- There is no additional benefit in adding sodium cromoglycate to an established regimen of ICS or systemic corticosteroid.1
- Sodium cromoglycate may be used as alternative to or in addition to SABA for the prevention of exercise-induced asthma.
- There is no clear evidence of benefit with sodium cromoglycate in children aged less than 5 years.5

Administration

Begin with 2 puffs 3–4 times daily, but administration twice daily is effective and more practical for maintenance.

- The CFC-free formulation is stickier than the original and the inhaler mouthpiece needs to be cleaned regularly to prevent blockage of the nozzle. It has two actuators in the pack to enable one to be used while the other is being cleaned.
- This information must be emphasised to patients, parents and carers. Full instructions are included in the consumer leaflet in each pack.

<table>
<thead>
<tr>
<th>DOSAGE</th>
<th>For frequent intermittent asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDI</td>
<td>1 mg/inhalation; 5 mg/inhalation (5 mg/inhalation MDI is preferred for most children)</td>
</tr>
<tr>
<td></td>
<td>1 mg/inhalation, 2–3 inhalations 2–3 times daily or 5 mg/inhalation, 1–3 inhalations 2–3 times daily, according to severity</td>
</tr>
</tbody>
</table>
Nedocromil sodium *(Tilade CFC-free)*

Nedocromil sodium is chemically distinct from both sodium cromoglycate and corticosteroids. It inhibits early and late-phase asthmatic reactions following allergen, exercise and osmotic challenge.

- **Nedocromil sodium is of some benefit in adults and children aged over 5 years.**
- In comparative studies with sodium cromoglycate, nedocromil has been shown to produce a similar protective effect against allergen and exercise, but has a longer duration. It may also be useful in seasonal allergic asthma.
- **Nedocromil has an effect on sensory nerves and therefore may be effective for the treatment of asthmatic cough. Reduction in cough may occur within 2–3 days of commencing the therapy.**
- **Adverse effects are infrequent and include headache, nausea, minor throat irritation and cough. Some patients may complain about the distinctive taste of nedocromil.**

**Indications**

- **Treatment of mild-moderate persistent asthma in adults and frequent episodic to mild persistent asthma in children over 2 years of age**
- **Prevention of exercise-induced asthma.**

**Administration**

The CFC-free formulation is stickier than the original and the inhaler mouthpiece needs to be cleaned regularly to prevent blockage of the nozzle. It has two actuators in the pack to enable one to be used while the other is being cleaned. This information must be emphasised to patients, parents and carers. Full instructions are included in the consumer leaflet in each pack.

**Dosage**

**MDI**

2 mg/inhalation

Commence with 2 inhalations 4 times daily and maintain for one month.

Once good symptom control and lung function improvement is achieved, the dose can usually be reduced to 2 inhalations twice daily.

**Anti-immunoglobulin therapy**

e.g. omalizumab (rhuMab-E25) *(Xolair)*

Omalizumab is a recombinant humanised monoclonal antibody to IgE. While ICS and leukotriene modifiers block the actions of inflammatory mediators, a newer approach in patients with allergic asthma is to prevent the inflammatory response at a higher point in the allergic cascade. Anti-immunoglobulin E (IgE) therapy prevents the release of inflammatory mediators (histamines, leukotrienes, cytokines and others) by blocking the interaction of allergens with IgE on the cell surface of mast cells and basophils.

- **Injection-site reactions can occur after the use of omalizumab.**
- A numerically higher (statistically non-significant) rate of malignancy has been recorded in patients receiving omalizumab, compared with control groups of patients with asthma, but a causal link has not been established.
- **This therapy is relatively expensive.**

**Indications**

Omalizumab is indicated for the management of moderate allergic asthma in adults and adolescents older than 12 years, who are using ICS and have elevated IgE levels.

Omalizumab achieves a modest reduction in ICS requirement. Omalizumab may be useful in patients with asthma that is poorly controlled despite the use of high-dose ICS, or patients who require frequent or long-term courses of systemic corticosteroids.

- When given in combination with ICS, omalizumab markedly reduces the risk of asthma exacerbations compared with placebo, but does not significantly reduce exacerbation rates in patients using oral corticosteroids.
Oral or parenteral corticosteroids
e.g. prednisolone, prednisone, panafcortelone (Panafcort)

Oral corticosteroids are established as the treatment of choice for severe exacerbations. These drugs are standard treatment for exacerbations that have become severe despite an increase in ICS or combination therapy. (For advice on initiating oral corticosteroid therapy, see Managing exacerbations)

DOSAGE
When given orally, a large initial dose should be used, e.g. for prednisone or prednisolone:
Adults:  40–60 mg
Children:  1 mg/kg up to 50 mg as a single daily dose.

In young children or those unable to swallow tablets, prednisolone is now available in liquid form (5 mg/mL Predmix, Redipred).

Symptom controllers
e.g. salmeterol (Serevent) (green inhaler); eformoterol (Foradile, Oxis) (pale blue inhaler)

Symptom controllers (LABAs) produce prolonged bronchodilation for up to 12 hours. They also protect the airways from bronchoconstriction secondary to exposure to allergens, non-specific stimuli or exercise. These agents are usually taken on a regular basis together with an ICS. The addition of a LABA to ICS improves lung function and symptoms and reduces exacerbations to a significantly greater degree than increasing the dose of ICS alone.

The addition of a LABA to ICS should be considered when:
• symptoms or sub-optimal lung function persist on ICS alone
• it is desirable to reduce the current dose of ICS while maintaining optimal asthma control.

If a patient fails to receive clinical benefit from a LABA after one month of treatment, the LABA should be withdrawn.

Adverse effects are similar in type and frequency to those of SABAs and include muscle tremor, headache and palpitations. A few patients may experience paradoxical bronchospasm as an immediate reaction to propellant in MDIs. Insomnia may occur.

The timing of adding a LABA to ICS should be individually tailored but is usually indicated if asthma control is not obtained at low-to-moderate doses of ICS alone. Inadequate symptom control from ICS should prompt a discussion about ICS use and adherence.

• Eformoterol has a rapid onset of action and can also be used as a reliever medication in adults.
• Salmeterol has a delayed onset of action and should not be used as reliever therapy.

Salmeterol (Serevent)
Salmeterol is currently available as a pressurised MDI and as a dry powder for inhalation via Accuhaler. It is also available in combination with fluticasone (see Combination medications).

DOSAGE
MDI  25 mcg/inhalation
Accuhaler  50 mcg/inhalation
Adults and children over 4 years: 50 mcg bd
In adults with severe airflow limitation:
Up to 100 mcg bd, although this dose is frequently associated with side effects.

Eformoterol (Foradile, Oxis)
Eformoterol is a LABA with a rapid onset of action. It is available in two dry powder devices: Aerolizer and Turbuhaler. It is also available in combination with budesonide (see Combination medications).

DOSAGE
Adults:
Aerolizer (Foradile) 12 mcg/inhalation:
1–2 capsules (12–24 mcg) bd. The total daily dose should not exceed 48 mcg
Turbuhaler (Oxis) 6 mcg and 12 mcg/inhalation: 6–12 mcg bd
Adults with more severe airflow limitation may require 24 mcg bd
Maximum daily dose 48 mcg
Eformoterol has a fast onset of action (1–3 minutes), and can therefore be used as reliever medication in asthma patients > 18 years, maximum recommended daily dose 72 mcg.

Children 5 years and over:
Aerolizer (Foradile) 12 mcg/inhalation: 1 capsule (12 mcg) bd
Maximum daily dose 24 mcg

Children 12 years and over:
Turbuhaler (Oxis) 6 mcg and 12 mcg/inhalation: 6–12 mcg bd
Maximum daily dose 24 mcg
Combination medications

Fluticasone and salmeterol (Seretide) (purple inhaler); budesonide and eformoterol (Symbicort) (red and white inhaler)

Using a fixed-dose combination inhaler – a single device containing both an ICS and LABA – is as effective as administering the ICS and LABA via separate inhalers. There is no evidence to suggest that the dose–response curve for ICS when given together with LABA differs from the known dose–response characteristics of ICS monotherapy.

The combination of LABA and ICS should be considered when:
- symptoms or sub-optimal lung function persist on ICS alone
- it is desirable to reduce the current dose of ICS while maintaining optimal asthma control
- initiating asthma treatment in a patient with moderate-to-severe asthma in whom rapid symptom improvement is needed.

Combination medications are available in both MDI and DPI forms. Comparison of the medication delivery between devices and resulting asthma control has produced similar results. However, individual variation in clinical response between devices may occur. Regardless of which type of device is considered to provide the best results, the choice of inhaler device for an individual should be based upon patient factors e.g. age, strength, dexterity, vision, cognition, inspiratory flow rate and the patient’s personal preference.

The use of combination ICS–LABA inhalers is well tolerated and there is no evidence that the adverse effects of either drug are potentiated by simultaneous delivery. Adverse effects are pharmacologically predictable, based on the beta-adrenergic activity of LABAs (tremor, tachycardia, palpitations and headache) and are no different when the drugs are administered in separate devices or together in one device.

Fluticasone and salmeterol (Seretide)

Seretide is delivered by CFC-free MDI and Accuhaler. The Accuhaler is suitable for patients who have good inspiratory flows but coordination difficulties when using MDIs.

**DOSAGE**

**MDI**
- 50/25: 50 mcg fluticasone and 25 mcg salmeterol
- 125/25: 125 mcg fluticasone and 25 mcg salmeterol
- 250/25: 250 mcg fluticasone and 25 mcg salmeterol

Each MDI contains 120 doses

**Accuhaler**
- 100/50: 100 mcg fluticasone and 50 mcg salmeterol
- 250/50: 250 mcg fluticasone and 50 mcg salmeterol
- 500/50: 500 mcg fluticasone and 50 mcg salmeterol

Each Accuhaler contains 60 doses

Adults and children > 12 years:
- Two inhalations bd of MDI 50, 125 or 250 depending on the patient’s asthma severity
- Children 4 years and over: Two inhalations bd of MDI 50

Adults and children > 12 years:
- One inhalation bd of Accuhaler 100, 250 or 500, depending on the patient’s asthma severity
- Children 4 years and over: One inhalation bd of Accuhaler 100

- Budesonide and eformoterol combination (Symbicort) has a rapid onset of action and can also be used as a reliever medication in adults.
- Fluticasone and salmeterol combination (Seretide) has a delayed onset of action and should not be used as reliever therapy.
**Budesonide and eformoterol (Symbicort)**

*Symbicort* is a combination medication containing budesonide and eformoterol. It is a dry powder delivered by *Turbuhaler*.

**PRESENTATION**

*Turbuhaler* 100/6:
- 100 mcg budesonide and 6 mcg eformoterol

*Turbuhaler* 200/6:
- 200 mcg budesonide and 6 mcg eformoterol

*Turbuhaler* 400/12:
- 400 mcg budesonide and 12 mcg eformoterol

Each *Turbuhaler* 100/6 contains 120 doses
Each *Turbuhaler* 200/6 contains 120 doses
Each *Turbuhaler* 400/12 contains 60 doses

**DOSAGE**

There are 2 alternative treatment regimens.

1) Maintenance therapy

*Symbicort Turbuhaler* 100/6 or 200/6:
- Adults and children >12 yrs: 1–2 inhalations of twice daily, max 4 inhalations/day
- If higher dose needed, use *Symbicort Turbuhaler* 400/12

*Symbicort Turbuhaler* 400/12:
- Adults ≥18 yrs needing high dose treatment: 2 inhalations twice daily; reduce to 1 inhalation twice daily when asthma controlled

2) Maintenance and reliever therapy

*Symbicort Turbuhaler* 100/6 or 200/6:
- Adults and children >12 years:
  - **Maintenance**: 1–2 inhalations twice daily or 2 inhalations once daily
  - **Reliever**: As needed in response to symptoms. No more than 6 inhalations should be taken on any one occasion. A total daily dose of more than 8 inhalations is normally not needed, however a total daily dose of up to 12 inhalations can be used temporarily.

The 400/12 strength should not be used for the *Symbicort* maintenance and reliever therapy regimen.

---

### Other medications and asthma

#### Antibiotics

- Antibiotics should be reserved for specific infections and are therefore rarely indicated in the treatment of asthma exacerbations.
- Mucus hypersecretion and a productive cough are a frequent manifestation of asthma. Discoloured sputum may be due to allergic inflammation and should not be interpreted as an indication of infection in the absence of other symptoms or signs.

#### Antihistamines

- Antihistamines may be used to treat associated nasal and other allergy symptoms.
- Older antihistamines are effective but also have significant anticholinergic effects, which can lead to sedation, reduced mental alertness and drying up of secretions. Less sedating antihistamines (cetirizine, fexofenadine, loratadine and desloratadine) have minimal or no anticholinergic effects and are the preferred option for most people.

#### Sedatives

- Sedatives are contraindicated during an acute asthma attack.
- Agitation during an attack may be due to bronchospasm and hypoxaemia and is better treated with beta₂ agonists and oxygen.
- Most sedatives, including benzodiazepines and zopiclone (and to a lesser degree, zolpidem), will blunt respiratory drive.

#### Medications that can exacerbate asthma

A person with asthma who is started on any new medication for another health condition should be advised to make sure they carry their reliever with them, and to watch for deterioration in their asthma. If deterioration occurs they should seek medical advice.

- Certain medications are known to trigger asthma. Others may adversely affect asthma control.
- All patients with asthma (and/or their carers) should be advised to ask their doctor or pharmacist about the effect a new medicine may have on their asthma.
- To facilitate the process, emphasise to people with asthma the benefits of continuing care by one doctor who will be aware of the patient’s asthma when prescribing other medications.
• It is also an advantage to have medications dispensed by the one pharmacist or pharmacy, as they may be aware of a person’s health conditions and lifestyle factors.

Medication-induced asthma can be separated into predictable and unpredictable/idiosyncratic asthma reactions. Predictable bronchoconstriction may occur with:
• beta blockers (used in the management of hypertension, cardiac disorders, migraine and glaucoma)
• cholinergic agents (e.g. carbachol, pilocarpine)
• cholinesterase inhibitors (e.g. pyridostigmine).

Unpredictable medication-induced asthma exacerbations may occur due to aspirin and other non-steroidal drugs (including cyclo-oxygenase (COX)-2 inhibitors) used for arthritis and inflammatory disorders.
• Be aware of the triad of nasal polyps, asthma and aspirin intolerance.
• An asthma exacerbation caused by NSAIDs is characterised by flushing and rhinorrhoea, often within a few minutes to an hour after administration.
• Other drugs that may cause reactions include carbemazepine and parenteral drugs: penicillin, iron dextran complex, hydrocortisone, ipratropium bromide, aminophylline, N-acetyl cysteine, and preservatives such as bisulfites, metabisulfites and benzalkonium chloride.

Delivery devices

Medications used to treat asthma are usually administered by inhalation. In terms of the benefit:harm ratio, inhaled drug delivery is superior to oral or parenteral delivery for SABAs, anticholinergics, LABAs and ICS.22
• Two different methods of inhalation are used:
  • Metered-dose inhaler (MDI) with or without the use of a spacer.
  • Dry-powder inhaler (DPI).
• Provided the devices are used correctly, there is no evidence of long-term clinical advantage of one device over another.23
• In general, patients with adequate inspiratory force and adequate hand-lung coordination can use either a DPI or an MDI. For older patients who have inadequate inspiratory force and/or poor coordination, use of an MDI with a spacer is preferred. Alternatively, a breath-activated MDI may warrant consideration.
• A DPI or an MDI used with a spacer may reduce the oropharyngeal disposition of medication and may reduce the local effects of ICS.22 However, there is no evidence that these devices reduce the systemic adverse effects of ICS, possibly because systemic absorption occurs as much through the bronchial circulation as it does through oral or gastrointestinal absorption.22

Deposition
• DPI: Deposition in the lung depends primarily on the inspiratory force. Both Turbuhaler and Accuhaler demonstrate acceptable drug delivery at inspiratory flow rates greater than 30 L/min.
• MDI: Deposition via MDI depends primarily on hand-lung coordination; the inspiratory force has no effect on either dosage delivery or average particle size. If adequate inhalation is not feasible, an MDI with a spacer is preferred. If inhalation is adequate, the patient’s inspiratory force and coordination determine the choice between an MDI (whether or not ‘breath-actuated’ or with a spacer) and a DPI. A rare side effect of metered-dose aerosols is bronchoconstriction.24

Prescribing devices
• When prescribing multiple medications, aim for consistency in the method of administration. MDIs with holding chambers produce outcomes at least equivalent to nebuliser delivery.25 Only in exceptional cases should oral beta2 agonist therapy or inhalation using an electric-powered jet nebuliser be considered.
• There is no evidence to dictate an order in which devices should be tested. In the absence of evidence, the most important points to consider are patient preference and local cost.
• In adults and children, patient preference and ability to use the device effectively should play a key role in the choice of delivery device (age is a major determinant in ability to use a device effectively). If the patient is unable to use a device satisfactorily an alternative should be found.
• The medication needs to be titrated against clinical response to ensure optimum efficacy.
• Inhaler technique should be reassessed as part of structured clinical review.
**Device training**

- It is essential that adults with asthma be competently trained in the correct technique of inhaler use. With good instruction, most adults are able to effectively use any of the commercially available devices. The device that best fits the needs and tolerability of the patient should be chosen.\(^{22}\)
- Before considering a higher dose of medication or the addition of another agent, reassess the inhaler technique and adherence. The technique should be reviewed regularly, especially if asthma is poorly controlled.\(^{22}\)

**Use and care of spacers**

- Spacers should be used:
  - by adults with poor coordination when using an MDI
  - by children of all ages: (with mask for those aged up to 2 years and mouthpiece for those aged over 2–5 years)
  - during an acute asthma attack.
- The spacer should be compatible with the MDI being used.
- The drug should be administered by repeated single actuations of the MDI inhaler into the spacer, each followed by inhalation.
- There should be minimal delay between MDI actuation and inhalation.
- Tidal breathing is as effective as single breaths.
- Spacers should be cleaned monthly rather than weekly as per manufacturer’s recommendations or performance is adversely affected. They should be washed in warm water with kitchen detergent and allowed to dry in air; drying with a cloth or paper towel can result in electrostatic charge (‘static’) on the inside of the spacer, which can reduce availability of dose. The mouthpiece should be wiped clean of detergent before use.
- Where spacers are for the use of more than one individual, guidelines for infection control should be consulted.
- Drug delivery may vary significantly due to static charge. Metal and other antistatic spacers have been shown to be effective in this respect.
- Spacers should be replaced at least every 12 months but some may need to be changed after six months of use.

**Practice tips**

- When using MDIs common side effects such as oropharyngeal thrush and hoarseness can be reduced by using a valved spacer and meticulous oral hygiene after each dose (rinse, gargle and spit).
- If coughing is a problem with the use of an MDI consider addition of a spacer or alternatively, use of an automatic breath-activated device such as Accuhaler, Autohaler or Turbuhaler.
- Breath-activated devices may be advantageous for those with a poor press-and-breathe technique.
- There is no evidence that any particular device reduces the risk of systemic adverse effects.

**Practice tips**

- Inhaled drug delivery is superior to oral (or parenteral) delivery for SABAs, anticholinergics, LABAs and ICS.
- There is no significant difference between delivery devices when used correctly.
- MDI plus large-volume spacer is at least as effective as a wet nebuliser in mild to moderate acute asthmatic episodes.
- People with asthma should receive adequate training in their inhaler technique to ensure competence.
- Device technique should be reassessed and reinforced frequently at appropriate opportunities.
- Choice of device should be made on the basis of ease of use, patient preference/suitability and overall cost.

---

**Devices for children**

<table>
<thead>
<tr>
<th>Medication Delivery</th>
<th>Under 2 Years</th>
<th>2–4 Years</th>
<th>5–7 Years</th>
<th>8 Years and older</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Route of administration</strong></td>
<td>MDI, small-volume spacer and mask</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td><strong>MDI, small-volume spacer and mouthpiece</strong></td>
<td></td>
<td></td>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Dry powder device</strong></td>
<td></td>
<td>Yes</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td><strong>Breath-activated device</strong></td>
<td></td>
<td>Possible</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td><strong>MDI and large-volume spacer</strong></td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MDI (alone)</strong></td>
<td>Yes</td>
<td></td>
<td></td>
<td>Yes (but spacer preferred especially for ICS)</td>
</tr>
</tbody>
</table>
• Some children in the 5–7 year age group are able to use dry powder devices effectively.
• Nebulisers can be used for children in any age group who are unable to comply with the above delivery devices. However, even in infants, spacers are the preferred and more efficient mode of delivery.
• For efficient drug delivery from a spacer, the device should be loaded with one puff at a time, and the child should take either 4 tidal breaths, or a single vital capacity breath.

**Beta₂ agonist delivery for acute asthma**

• MDI + spacer is at least as good as a nebuliser for treating mild and moderate exacerbations of asthma in adults and in children aged two years and older.²⁵,²⁷,²⁸
• Doses should be titrated according to clinical response. There are no data to make recommendations for children under the age of two or in patients with severe (life-threatening) asthma.

**Beta₂ agonist delivery for stable asthma**

• For children aged 0–5 years, MDI + spacer (with mask for those aged up to 2 years and mouthpiece for those aged over 2 years) is the preferred method of delivery. Where this is ineffective a nebuliser may be required.
• In children aged 5–12 years, there is no significant difference between MDI and DPI, although administration via a spacer is recommended.
• In adults, there is no significant difference between MDI + spacer and DPI. (The lower pulse rate with MDI versus Turbuhaler is the only difference with regard to side effects.)²³,²⁹
• Choice of reliever inhaler for stable asthma should be based on patient preference and assessment of correct use. Many patients will not be prepared to carry a spacer.

**ICS delivery for stable asthma**

• For children aged 0–5 years, MDI + spacer is the preferred method of delivery. A face-mask is required until the child can breathe reproducibly using the spacer mouthpiece. Where this is ineffective a nebuliser may be required.
• There are no comparative data on delivery of ICS for stable asthma in children under 5 years.
• In children aged 5–12 years, MDI + spacer is as effective as DPI.²³,³⁰
• In adults, there is no clinical difference in effectiveness of MDI ± spacer versus DPI. A breath-actuated MDI is as effective as an MDI.
• Nebulisers have not been shown to be superior to MDI + spacer for delivery of ICS in chronic asthma. A specialised, specific nebuliser may provide improved lung function and reduced rescue therapy use, but at high prescribed doses. Higher doses (> 2 mg) are generally only licensed for use from a nebuliser.²³,³⁰

**Combination devices**

• Combination dry powder devices have similar efficacy as giving the same medications via separate devices.²¹,²⁶
• Combination dry powder devices may be more convenient, but titration of the dose requires consideration of the type of device and drug combination being used.
This chapter deals with the management of acute asthma in the hospital or emergency facility setting. For information on community-based management of acute or subacute worsening of symptom control and lung function, see Managing exacerbations.

### Managing acute asthma in adults

Table 1 summarises steps in the emergency care of an adult with acute asthma.

#### Practice points

- If the patient is acutely distressed, give oxygen and SABA immediately after taking a brief history and physical examination. ✓
- Assess response to treatment using spirometry, oxygen saturation, heart rate, respiratory rate and pulsus paradoxus status. ✓
- Wheeze is an unreliable indicator of the severity of an asthma attack and may be absent in severe asthma. ✓
- Ensure every patient receives adequate follow-up after an acute asthma episode, including review of medications, triggers and asthma action plan. ✓

#### Initial assessment

The initial assessment of an adult with acute asthma is summarised in Table 2.

- Spirometry is the lung function test of choice for assessing asthma severity during an acute episode (if the patient is able to perform the manoeuvre), and for monitoring the response to treatment.
- Patients who are acutely distressed require immediate oxygen and short-acting beta₂ agonist (SABA) before completing a full assessment.

### Managing acute asthma in children

If the patient is acutely distressed, give oxygen and SABA immediately after taking a brief history and physical examination.

- Emergency management of acute asthma in a child is based on initial administration of salbutamol 4–6 puffs (< 6 years) or 8–12 puffs (≥ 6 years) via MDI. I
- Load the spacer with one puff at a time and give each puff separately. III-1
- If treatment with an oral corticosteroid (e.g. prednisolone 1 mg/kg up to 60 mg as a single daily dose) has been initiated for a moderate-to-severe acute episode, continue for up to 5 days. I
Table 1. Summary of steps in emergency care of an adult with acute asthma

1. Take a brief history and perform a rapid physical examination before beginning treatment.
2. If the patient is acutely distressed, give oxygen and SABA immediately. Consider whether adrenaline is indicated. Oxygen therapy may be associated with respiratory depression and arrest in patients with chronic CO₂ retention, particularly those with chronic obstructive pulmonary disease (COPD).
3. Take a more detailed history and complete the physical examination. Initiate treatment with other agents, including systemic corticosteroids, as indicated. Further treatment depends on the severity of the episode and response to initial treatment (Table 3).
4. Perform spirometry and/or peak expiratory flow (PEF) measurement as soon as possible to gain an objective measure of airflow limitation.
5. Assess progress by continued close monitoring of objective measures of improvement. Spirometry is the most reliable measure of response to treatment. Measurement of PEF may be used if a spirometer is not available.
   • In adults with severe acute asthma, measure arterial blood gases after initiating treatment to assess CO₂ retention as well as to enable management of hypoxaemia.
   • Keep oxygen saturation on oximetry (SaO₂) above 90%.
   • Heart rate, respiratory rate and pulsus paradoxus (abnormal decrease in systolic blood pressure during inspiration) are also useful measures of response.
   • Reduction in wheezing is an unreliable indicator of improvement, as it may indicate deterioration.
   • Intubation and ventilation are indicated for patients with acute respiratory failure that does not respond to treatment and for respiratory arrest or exhaustion suggesting impending respiratory arrest.

Table 2. Initial assessment of acute asthma in adults

<table>
<thead>
<tr>
<th>Findings</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe and life threatening*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical exhaustion</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Paradoxical chest wall movement may be present</td>
</tr>
<tr>
<td>Talks in</td>
<td>Sentences</td>
<td>Phrases</td>
<td>Words</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>&lt; 100/min</td>
<td>100–120/min</td>
<td>More than 120/min†</td>
</tr>
<tr>
<td>Pulsus paradoxus</td>
<td>Not palpable</td>
<td>May be palpable</td>
<td>Palpable‡</td>
</tr>
<tr>
<td>Central cyanosis</td>
<td>Absent</td>
<td>May be present</td>
<td>Likely to be present</td>
</tr>
<tr>
<td>Wheeze intensity</td>
<td>Variable</td>
<td>Moderate to loud</td>
<td>Often quiet</td>
</tr>
<tr>
<td>PEF</td>
<td>More than 75% predicted (or best if known)</td>
<td>50–75% predicted (or best if known)</td>
<td>Less than 50% predicted (or best if known) or less than 100 L per min#</td>
</tr>
<tr>
<td>FEV₁</td>
<td>More than 75% predicted</td>
<td>50–75% predicted</td>
<td>Less than 50% predicted or less than 1 L</td>
</tr>
<tr>
<td>Oximetry on presentation</td>
<td></td>
<td></td>
<td>Less than 90% Cyanosis may be present**</td>
</tr>
<tr>
<td>Arterial blood gases</td>
<td>Not necessary</td>
<td>Necessary if initial</td>
<td>Necessary††</td>
</tr>
<tr>
<td>(assay)</td>
<td></td>
<td>response poor</td>
<td></td>
</tr>
<tr>
<td>Other investigations</td>
<td>Not required</td>
<td>May be required</td>
<td>Check for hypokalaemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chest X-ray to exclude other pathology (e.g. infection, pneumothorax)</td>
</tr>
</tbody>
</table>

*Any of these features indicates that the episode is severe. The absence of any feature does not exclude a severe attack.
†Bradycardia may be seen when respiratory arrest is imminent.
‡Paradoxical pulse is more reliable in severe obstruction. Its presence (especially if > 12 mmHg) can identify patients who need admission. Absence in those with severe exacerbations suggests respiratory muscle fatigue.
*Patient may be incapable of performing test.
** Measurement of oxygen saturation is required: many patients look well clinically and may not appear cyanosed despite desaturation.
††PaCO₂ > 50 mmHg indicates respiratory failure. PaO₂ < 60 mmHg indicates respiratory failure.
**History**

The following information should be obtained as soon as possible after the patient presents with acute asthma:

- the cause of the present exacerbation (e.g. upper respiratory tract infection, allergen exposure, food allergy)
- the duration of symptoms. (With increasing duration of the attack, exhaustion and muscle fatigue may precipitate ventilatory failure.)
- the severity of symptoms, including exercise limitation and sleep disturbance
- details of all current asthma medications, doses and amounts used, including the time of the last dose. (Distinguish between preventer medications and reliever medications used for the acute attack.)
- medication adherence history
- details of other medications. (Check whether the person has used any medications that might aggravate asthma, including complementary/herbal medications.)
- prior hospitalisations and Emergency Department visits for asthma or anaphylaxis, particularly within the last year
- exposure to nonsteroidal anti-inflammatory drugs or aspirin
- prior episodes of severe life-threatening asthma (e.g. admissions to intensive care unit, ventilation)
- significant coexisting cardiopulmonary disease
- the presence of underlying chronic obstructive pulmonary disease (indicates risk of CO\textsubscript{2} retention)
- known immediate hypersensitivity to food, bee sting or drugs
- smoking history.

**Management**

The initial management of adults with acute asthma is summarised in Table 3. For information on the assessment of severity, see Table 2, Initial assessment of acute asthma in adults.

Patients with moderate-to-severe acute asthma require admission to hospital (consider intensive care) and continuous observation.

In adults with mild acute asthma, admission to hospital is usually not necessary. Observe patients for 1 hour after the episode is controlled to ensure full recovery. For information on community-based management of acute or subacute deterioration in symptom control and lung function, see Managing exacerbations.

---

**Give SABA via MDI plus spacer immediately**

Initially give 8–12 puffs salbutamol (100 mcg/dose) via MDI and spacer. Repeat as necessary (e.g. repeat every 15–30 minutes in a severe episode, 1–4 hours after the first dose in a moderately severe episode).

The use of SABAs by intermittent inhalation via MDI plus spacer is now recommended in the management of acute asthma, whether mild, moderate or severe.

- Delivery of SABA via MDI and spacer is equally effective as nebulisation in patients with moderate-to-severe acute asthma, other than those with life-threatening asthma (e.g. patients requiring ventilation).\(^1\) In patients who can inhale well enough to use an MDI, the use of IV SABA gives no advantage over inhaled treatment.
- Continuous nebulisation and intravenous therapy are alternatives in severe asthma. However, adverse events are more frequent.\(^2\,^3\,^4\)
- Use a nebuliser instead if the person cannot inhale adequately: a 5 mg nebul of salbutamol with 2 mL saline or 1 mL of salbutamol solution (5 mg/mL) with 3 mL saline as needed. If available give wall oxygen at a flow of 8–10 L/min. A mouthpiece delivers considerably more drug to the lung than a face-mask.\(^5\)
- If no response to SABA via inhaler or nebuliser, give salbutamol 250 mcg IV bolus then 5–10 mcg/kg/hour by IV infusion.

**Practice tip**

Salbutamol 8–12 puffs via MDI (100 mcg/dose) is equivalent to 5 mg via nebuliser. Alternatives are:
- terbutaline (Bricanyl) 500 mcg/dose 4–6 puffs
- eformoterol (Oxis Turbuhaler) 6 or 12 mcg/dose can be used. Up to 48 mcg in divided doses over 30 mins has been shown to be safe and effective.\(^6\)

**Start systemic corticosteroids**

All patients with moderate–severe acute asthma will require a course of systemic corticosteroids in addition to inhaled corticosteroids (ICS).

- Commence a short course of oral corticosteroids (e.g. prednisolone 0.5–1.0 mg/kg for 7–10 days)
- Alternatively, corticosteroids can be given IV: hydrocortisone 100 mg 6 hourly or 40–120 mg methylprednisolone once daily or 4–12 mg dexamethasone once daily. There is no significant advantage to using more than 400 mg hydrocortisone
per day (200 mg per day is adequate for most patients). Oral corticosteroids can be substituted when oral intake resumes.

ICS should be continued, but it is not clear whether this provides any additional benefit over systemic corticosteroids alone.

The roles of other agents in acute asthma care in adults

- Nebulised ipratropium bromide given in addition to SABA may improve bronchodilation. If using nebulised SABA, add ipratropium bromide 2 mL 0.05% (1 mg) with salbutamol 2 hourly.
- Aminophylline 25 mg/mL IV (6 mg/kg IV slow injection then 0.3–0.6 mg/kg/hour infusion) can be used as an alternative to IV salbutamol when an acute episode does not respond to inhaled SABA. However, the use of intravenous aminophylline is unlikely to provide a significant benefit in addition to therapeutically effective SABAs (i.e. where not compromised by concurrent use of beta blockers), and may increase adverse effects including nausea and vomiting.

Injection rate should not exceed 25 mg/minute to reduce the risk of hypotension, seizures and arrhythmia. Serum levels should be monitored for both maximal effect and toxicity.

- Magnesium sulphate (via nebuliser or IV, as available) can be added to improve airflow, although the evidence to support this is not strong. Suggested doses are 1.2–2 mg of MgSO₄ IV over 20 min or 2.5 mL isotonic MgSO₄ (250 mmol/L) by nebuliser.
- Adrenaline is required for respiratory arrest or exhaustion suggesting impending respiratory arrest. Give 5 mL of 1:10,000 solution slowly IV. An alternative is 0.5 mL of 1:1,000 (0.5 mg) solution IM, but IV is the preferred route due to unpredictable absorption and the possible need for another injection with IM administration.

Other investigations

Arrange chest X-ray if there is no response to initial therapy, if focal signs are present or if pneumothorax is suspected.

Check for hypokalaemia and correct if present.

Follow-up care after an acute asthma episode

- Ensure every patient receives adequate follow-up after an acute asthma episode, including review of medications, triggers and asthma action plan.

This is a valuable opportunity to review the patient's overall asthma management. Review of maintenance medications and asthma control is necessary e.g:

- Was previous baseline asthma control adequate?
- Is the patient's asthma action plan up to date?

Follow-up care is crucial for those who do not require hospitalisation:

- Identify trigger factors.
- Provide a written asthma action plan for the patient and their carers.
- Recommend rapid-onset beta₂ agonists as required for symptom control.
- Use spirometry to monitor lung function and reassess the treatment plan as necessary.
- Consider adding a LABA if patients are not already taking ICS–LABA combination therapy.
- Re-evaluate ICS dose and back-titrate at next review.

Patients who are hospitalised will require follow-up care on discharge, including:

- discharge summary to the patient’s usual GP
- appointment with the patient’s usual GP within 1 week
- ± outpatient department appointment with a consultant physician in 2 weeks
- interim written asthma action plan.

For more information about long-term asthma management, see Ongoing care.
**Table 3. Initial management of adults with acute asthma**

<table>
<thead>
<tr>
<th><strong>Treatment</strong></th>
<th><strong>Mild episode</strong></th>
<th><strong>Moderate episode</strong></th>
<th><strong>Severe episode</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital admission</td>
<td>Probably not necessary</td>
<td>Admit</td>
<td>Admit. Consider admission to intensive care unit.</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Flow rate adjusted to achieve SaO₂ &gt; 90%. Frequent measurement of arterial blood gases is indicated in severe asthma and those not responding to treatment.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SABA via MDI + spacer</td>
<td>8–12 puffs salbutamol</td>
<td>8–12 puffs salbutamol every 1–4 hours</td>
<td>8–12 puffs salbutamol every 15–30 mins</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **SABA nebulised, e.g. salbutamol or terbutaline, with O₂ 8 L/min** | One salbutamol 5 mg/2.5 mL nebule  
One terbutaline 5 mg/2 mL respule  
Salbutamol 1 mL of 5 mg/mL solution + 3 mL saline | One salbutamol 5 mg/2.5 mL nebule  
One terbutaline 5 mg/2 mL respule  
Salbutamol 1 mL of 5 mg/mL solution + 3 mL saline | Salbutamol 1 mL of 5 mg/mL solution + 3 mL saline every 15–30 mins |
|                               | or                |                          | If no response, give salbutamol 250 mcg (0.5 mL of 500 mcg/mL solution) IV bolus over one minute then IV 5–10 mcg/kg/hour |
| Nebulised ipratropium bromide  | Not necessary     | Optional              | Ipratropium bromide 2 mL 0.05% (500 mcg) with salbutamol 2 hourly |
| Oral corticosteroids e.g. prednisolone | Yes (consider) | Yes 0.5–1.0 mg/kg initially | Yes 0.5–1.0 mg/kg initially |
| Intravenous steroids e.g. hydrocortisone (or equivalent) | Not necessary | ¹Hydrocortisone 250 mg (or equivalent) | ²Hydrocortisone 250 mg 6 hourly for 24 hours then review |
| Theophylline/ aminophylline     |                   |                      | #Aminophylline 25 mg/mL: give 6 mg/kg slow IV injection then 0.3–0.6 mg/kg/hour IV infusion |
| Adrenaline                     | Not indicated     | Not indicated         | 5 mL of 1:10,000 solution slowly IV if anaphylaxis present |
| Chest X-ray                    | Not necessary unless focal signs present | Not necessary unless focal signs present, or no improvement to initial treatment | Necessary if no response to initial therapy or pneumothorax suspected |
| Observations                   | Regular           | Continuous            | Continuous         |
| Other                          |                   |                      | Treat for hypokalaemia if present |

¹SABA via MDI and spacer is as effective as nebulisation in patients with moderate-to-severe acute asthma, other than those with life-threatening asthma (e.g. patients requiring ventilation).¹

²Use IV corticosteroids in moderate acute asthma if oral route not convenient

³Either oral or IV corticosteroids can be given initially. Follow with oral course.

* Can be given as an alternative to IV salbutamol
Managing acute asthma in children

Table 4 summarises steps in the emergency care of a child with acute asthma.

**Practice points**

- If the patient is acutely distressed, give oxygen and SABA immediately after taking a brief history and physical examination.
- Emergency management of acute asthma in a child is based on initial administration of salbutamol 4–6 puffs (< 6 years) or 8–12 puffs (≥ 6 years) via MDI. (I)
- Load the spacer with one puff at a time and give each puff separately. (III-1)
- If a course of oral corticosteroids (e.g. prednisolone 1 mg/kg up to 60 mg as single daily dose) has been initiated for a moderate-to-severe acute episode, continue for up to 5 days. (I)

**Initial assessment**

The initial assessment of a child with acute asthma is summarised in Table 5.

Children who are acutely distressed require immediate oxygen and short-acting beta2 agonist (SABA) before completing a full assessment.

**Management**

The management of acute episodes is based on salbutamol delivered via MDI and spacer, repeated at 20-minute intervals until control is achieved (Table 6).

Nebulised salbutamol is reserved for life-threatening episodes. For dose-equivalence information on nebulisers and MDIs see Table 7. Salbutamol dose equivalents.

Table 4. Summary of steps in emergency care of a child with acute asthma

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Take a brief history and perform a physical examination (Table 5).</td>
</tr>
<tr>
<td>2.</td>
<td>Give salbutamol via MDI plus spacer immediately: usually 6 puffs for children under 6 years and 12 puffs for children 6 years and over. If the patient is acutely distressed, give immediately (Table 6).</td>
</tr>
<tr>
<td></td>
<td>- The MDI used with a spacer has replaced the nebuliser as the standard method of delivering SABAs in hospital-based care of children with acute asthma (Table 6).</td>
</tr>
<tr>
<td></td>
<td>- Each puff is given separately: the spacer should only be loaded with one puff at a time.</td>
</tr>
<tr>
<td></td>
<td>- The total dose (up to 12 puffs) is based on medical assessment in addition to the child’s age. With experience, parents learn to judge SABA dose requirement.</td>
</tr>
<tr>
<td></td>
<td>3. Complete a full assessment and initiate other treatment, including systemic corticosteroids and oxygen as indicated (Table 6).</td>
</tr>
<tr>
<td></td>
<td>- Adrenaline may be indicated if asthma occurs as part of an anaphylactic reaction, depending on severity.</td>
</tr>
<tr>
<td></td>
<td>- There is now convincing evidence that intravenous magnesium sulphate provides additional benefit in children with severe asthma treated with bronchodilators and corticosteroids. Magnesium sulphate has an excellent safety profile and its place in the management of acute severe asthma is similar to that of aminophylline.</td>
</tr>
<tr>
<td></td>
<td>4. Closely monitor response to treatment and repeat SABA as indicated (Table 6).</td>
</tr>
</tbody>
</table>

Managing a severe acute asthma episode in a child

Arrange for admission to hospital and consider intensive care.

- Initially, give salbutamol 6 puffs (children under 6 years) or 12 puffs (children 6 years and over) every 20 mins for the first hour (i.e. three doses).
- If the episode appears to be life threatening, use continuous nebulised salbutamol.
- If no response, give salbutamol 15 mcg/kg IV over 10 mins, then 1 mcg/kg/min infusion.
- Give supplementary oxygen and monitor SaO2 by oximetry. Arterial blood gases may also be required.
- Give systemic corticosteroids, either:
  - Begin a course of oral prednisolone (1 mg/kg/dose daily up to 60 mg for up to 5 days); or
  - Give methylprednisolone IV 1 mg/kg up to 60 mg every 6 hours on Day 1, then every 12 hours on Day 2, then daily.
- Give ipratropium 2 puffs (children under 6 years) or 4 puffs (children 6 years and over) every 20 minutes for the first hour (i.e. three doses). Nebulised ipratropium may be used as an alternative.
- Give magnesium sulphate 50% 0.1 ml/kg (50 mg/kg) IV over 20 minutes, then 0.06 ml/kg/hr (30 mg/kg/hr). Target serum magnesium 1.5–2.5 mmol/L. |
- Aminophylline, if used, should only be given in an intensive care unit. Give a loading dose of 10 mg/kg then a maintenance dose of 1.1 mg/kg/hour (children under 9 years old) or 0.7 mg/kg/hour (children 9 years and over).
- Arrange chest X-ray if there is no response to initial therapy or if pneumothorax is suspected.
Managing a moderate acute asthma episode in a child

Children with moderate acute asthma may require hospital admission.
- Initially, give salbutamol 6 puffs (children under 6 years) or 12 puffs (children 6 years and over).
- If initial response is inadequate, repeat at 20-minute intervals for two further doses, then give every 1 to 4 hours.
- Monitor oxygen saturation using oximetry. Supplemental oxygen may be required.
- Begin a course of oral prednisolone 1 mg/kg daily for up to 3 days.
- Chest X-ray is not necessary unless focal signs are present.
- If the child is not admitted to hospital, observe for at least 1 hour after the last dose of medication.

Managing a mild acute asthma episode in a child

Initially, give salbutamol 6 puffs (children under 6 years) or 12 puffs (children 6 years and over).
- Review response after 20 minutes and repeat if necessary as for moderate acute episodes.
- Consider beginning a short course of oral corticosteroids (prednisolone 1 mg/kg daily for up to 3 days).
- Observe for at least 20 minutes after the last dose before allowing the child to go home.

Table 5. Initial assessment of acute asthma in children

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe and life-threatening*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Altered consciousness</td>
<td>No</td>
<td>No</td>
<td>Agitated</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Confused/drowsy</td>
</tr>
<tr>
<td>Oximetry on presentation (SaO₂)</td>
<td>94%</td>
<td>94–90%</td>
<td>Less than 90%</td>
</tr>
<tr>
<td>Talks in</td>
<td>Sentences</td>
<td>Phrases</td>
<td>Words</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Unable to speak</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>Less than 100 beats/min</td>
<td>100–200 beats/min</td>
<td>More than 200 beats/min</td>
</tr>
<tr>
<td>Central cyanosis</td>
<td>Absent</td>
<td>Absent</td>
<td>Likely to be present</td>
</tr>
<tr>
<td>Wheeze intensity</td>
<td>Variable</td>
<td>Moderate to loud</td>
<td>Often quiet</td>
</tr>
<tr>
<td>PEF**</td>
<td>More than 60% predicted or personal best</td>
<td>40–60% predicted or personal best</td>
<td>Less than 40% predicted or personal best</td>
</tr>
<tr>
<td>FEV₁</td>
<td>More than 60% predicted</td>
<td>40–60% predicted</td>
<td>Less than 40% predicted</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Unable to perform</td>
</tr>
</tbody>
</table>

*Any of these features indicates that the episode is severe. The absence of any feature does not exclude a severe attack.

**Children under 7 years old are unlikely to perform PEF or spirometry reliably during an acute episode. These tests are usually not used in the assessment of acute asthma in children.
Community-based first aid

A simplified “4 x 4 x 4” protocol has been developed for use in first aid where SABAs are the only available treatment. The protocol is safe and easy to follow, and is based on a gradual dose accumulation to a maximum of 12 puffs of salbutamol: Four puffs reliever, one puff at a time, with four breaths after each puff. Wait four minutes, then repeat. See First Aid for Asthma in Appendices.

Copies of the First Aid for Asthma chart are available from the National Asthma Council Australia and the Asthma Foundations.

Follow-up care after an acute asthma episode

- Give further SABA doses as needed, up to 3–4 hourly
- Give a short course of oral corticosteroids (e.g. prednisolone 1 mg/kg as single daily dose for up to 5 days). In children taking high-dose ICS it may be necessary to taper the dose over 3–5 days before ceasing.
- Provide clear instructions about when to return if asthma worsens. See Asthma action plans.
- Arrange follow-up appointment with regular practitioner to review overall management within 2 weeks.

For more information about long-term asthma management, see Ongoing care.
### Table 6. Initial management of children with acute asthma

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mild episode</th>
<th>Moderate episode</th>
<th>Severe and life-threatening episode</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital admission necessary</td>
<td>Probably not</td>
<td>Probably</td>
<td>Yes: consider intensive care</td>
</tr>
<tr>
<td>Supplementary oxygen</td>
<td>Probably not required</td>
<td>May be required. Monitor SaO₂</td>
<td>Required. Monitor SaO₂. Arterial blood gases may be required.</td>
</tr>
<tr>
<td>Salbutamol*</td>
<td>4–6 puffs (under 6 years) or 8–12 puffs (6 years or over). Review in 20 mins</td>
<td>6 puffs (under 6 years) or 12 puffs (6 years or over). If initial response inadequate, repeat at 20-minute intervals for two further doses. Then give every 1–4 hours.</td>
<td>6 puffs (under 6 years) or 12 puffs (6 years or over) every 20 mins for three doses in first hour. If life-threatening episode, use continuous nebulised salbutamol. If no response, bolus IV salbutamol 15 mcg/kg over 10 mins then 1 mcg/kg/min thereafter.</td>
</tr>
<tr>
<td>Ipratropium</td>
<td>Not necessary</td>
<td>Optional</td>
<td>2 puffs (under 6 years) or 4 puffs (6 years or over) every 20 minutes x 3 doses in first hour or nebulised ipratropium</td>
</tr>
<tr>
<td>Systemic corticosteroids</td>
<td>Yes (consider)</td>
<td>Oral prednisolone 1 mg/kg daily for up to 3 days</td>
<td>Oral prednisolone 1 mg/kg/dose daily for up to 5 days Methyiprednisolone IV 1 mg/kg 6 hourly on Day 1, 12 hourly on Day 2 then daily</td>
</tr>
<tr>
<td>Magnesium</td>
<td>No</td>
<td>No</td>
<td>Magnesium sulphate 50% 0.1 mL/kg (50 mg/kg) IV over 20 mins then 0.06 mL/kg/hr (30 mg/kg/hr): target serum Mg 1.5–2.5 mmol/L</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>No</td>
<td>No</td>
<td>Only in Intensive Care: loading dose 10 mg/kg. Maintenance 1.1 mg/kg/hour if under 9 years or 0.7 mg/kg/hour if 9 years and over</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>Not necessary unless focal signs present</td>
<td>Not necessary unless focal signs present</td>
<td>Necessary if no response to initial therapy or pneumothorax is suspected</td>
</tr>
<tr>
<td>Observations</td>
<td>Observe for 20 mins after dose</td>
<td>Observe for 1 hour after last dose</td>
<td>Arrange for admission to hospital</td>
</tr>
</tbody>
</table>

*In children with severe acute asthma that does not respond to initial treatment with inhaled SABA, bolus IV salbutamol 15 mcg/kg over 10 mins is effective and can avoid the need for continuous IV salbutamol and ICU admission.16,17

### Table 7. Salbutamol dose equivalents

<table>
<thead>
<tr>
<th>Salbutamol via MDI and spacer</th>
<th>is equivalent to</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 puffs (children &lt; 6 years)</td>
<td>2.5 mg nebulé</td>
</tr>
<tr>
<td>12 puffs (children ≥ 6 years)</td>
<td>5 mg nebulé</td>
</tr>
</tbody>
</table>
MANAGING EXACERBATIONS

SUMMARY OF PRACTICE POINTS | LEVEL OF EVIDENCE
--- | ---
**Management of exacerbations in adults**
A short (7–10 days) course of oral corticosteroids is the current standard treatment for adults with moderate-to-severe asthma exacerbations. | I
When administering a SABA via MDI during an exacerbation, use a spacer. | I
Merely doubling the maintenance ICS dose is not effective in managing exacerbations. | II
**Management of exacerbations in children**
A short (up to 5 days) course of oral corticosteroids (prednisolone 1 mg/kg up to 60 mg daily) is the current standard treatment for severe exacerbations. Closely monitor response to treatment. | I
Children who are taking regular preventive medication should continue taking the same dose during an exacerbation. | II
When administering a SABA via MDI during an exacerbation, use a spacer. | III-1
Merely doubling the maintenance ICS dose is not effective in managing exacerbations in children. | II

This chapter deals with the community-based management of asthma exacerbations. For information on managing acute asthma (including asthma emergencies) in the hospital setting, see Acute asthma.

The term "exacerbation" means an acute or subacute deterioration in symptom control and lung function (compared with the individual's usual level of variation) that is sufficient to cause distress or risk to health and requires a change in treatment.¹

The stage at which patients will present for additional treatment depends not only on the severity of symptoms, magnitude of reduction in lung function and the impact on daily activities, but also on access to care, costs, previous experience of health care and psychosocial factors including the patient's or carer's health-related beliefs.

- Fear of oral corticosteroids can delay presentation to medical services and therefore increase risk associated with an exacerbation.²
- Levels of anxiety and fear significantly influence patients' requests for asthma treatment. See Asthma and mental illness.

All patients and carers should be offered a current written asthma action plan to help them:

- make appropriate treatment adjustments in response to worsening asthma
- know when to use medical services (primary care or emergency facility).

See Asthma action plans.

---

¹Notes: Guidelines for detecting and managing asthma exacerbations are currently limited by the lack of standardised criteria for exacerbations, and this is being addressed by a European Respiratory Society (ERS)/American Thoracic Society (ATS) Task Force. There is limited clinical evidence available to guide the management of early or suspected exacerbations. Few studies have compared different treatment approaches (e.g. oral corticosteroids versus high-dose ICS) in a group of clinically homogenous patients experiencing comparable exacerbations of a defined severity level or stage of onset. Most of the evidence relevant to the treatment of exacerbations in patients with stable asthma is derived from clinical trials designed to evaluate various clinical parameters as action triggers for the initiation of extra treatment. These trials have used initiation of oral corticosteroids (with or without hospitalisation) as the endpoint, and many have defined "severe exacerbation" as the requirement for oral corticosteroids, based on the treating clinician's assessment.

---

*Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.*
Distinguish exacerbations from poor asthma control

Try to distinguish between ongoing poor asthma control and exacerbations. In patients with stable asthma, intermittent exacerbations (particularly those due to upper respiratory viral infections) will occur despite ongoing good asthma control overall. 

Inadequate asthma control – whether due to inappropriate drug treatment, poor adherence to treatment or incorrect inhaler technique – must be identified so that the treatment regimen and asthma action plan can be appropriately altered to achieve good asthma control. For more information, see Troubleshooting.

The role of PEF monitoring in detecting exacerbations

Practice tip
Monitoring of asthma control based on PEF may have the greatest benefit in patients who cannot readily perceive symptoms of airflow limitation.

Self-monitoring based on peak expiratory flow (PEF) might help some patients detect the onset of potentially severe exacerbations earlier, but there is no strong evidence that this offers any advantage over symptom monitoring for most patients. 

- Those who cannot easily perceive worsening airflow limitation or confuse symptoms (e.g. elderly patients) may benefit most from PEF-based monitoring. See Asthma in the elderly

- Before a 30% reduction in PEF from personal best is detected, most patients become aware of symptoms suggesting worsening asthma control (e.g. breathlessness, increased reliever usage) and either seek medical care or start additional treatment.

- Among patients using PEF-based asthma action plans to manage their asthma in response to fluctuations in symptom control, alterations in treatment should be based on deviation from personal best PEF, rather than comparison with predicted PEF.

- In children, no clear benefit has been demonstrated for PEF-based asthma action plans over symptom-based action plans.

For information on self-management in response to changes in asthma control, see Asthma action plans.

Managing asthma exacerbations in adults

Practice points

- A short (7–10 days) course of oral corticosteroids is the current standard treatment for adults with moderate-to-severe asthma exacerbations. (I)
- When administering a SABA via MDI during an exacerbation, use a spacer. (II)
- Merely doubling the maintenance ICS dose is not effective in managing exacerbations. (II)

Spirometry is the lung function test of choice for assessing asthma control during an acute or subacute episode or in response to treatment.

Oral corticosteroids

A short course of oral corticosteroids (e.g. prednisolone 0.5–1.0 mg/kg once daily for 7–10 days, depending on severity) is the current standard treatment for exacerbations including those presenting to emergency departments and those that have not resolved despite an increase in ICS or combination therapy.

- Oral corticosteroids are as effective as intramuscular corticosteroids.
- There is no evidence to suggest that prolonging the course beyond 10 days is indicated.
- Oral corticosteroids can be ceased abruptly at the end of the course. Tapering the dose does not prevent relapse and is not necessary after a short course.

Short-acting beta\textsubscript{2} agonists

There is strong evidence that inhaled short-acting beta\textsubscript{2} agonists (SABAs) are effective in the management of exacerbations.

- During exacerbations, SABAs should be administered via metered dose inhaler (MDI) and spacer.
- Delivery of SABA via MDI and spacer is equally effective as nebulisation in patients with moderate-to-severe acute asthma.
- Doses are as for acute asthma episodes: 8–12 puffs salbutamol 100 mcg/dose (or equivalent) via MDI, repeated after 1–4 hours or as necessary until control is achieved.
Managing exacerbations

For more information on SABA doses and administration of SABAs and the rapid-onset long-acting beta₂ agonist eformoterol during an exacerbation, see Acute asthma.

**Inhaled corticosteroids**

In adults with exacerbations not considered severe enough for admission to hospital, high-dose inhaled corticosteroids (ICS) for 1–2 weeks (e.g. FP 2000 mcg/day or BUD 3200 mcg/day) may be effective in controlling the episode, but it is not known whether ICS alone are as effective as a standard course of oral corticosteroids.

Merely doubling the maintenance ICS dose is **not effective** in managing exacerbations and higher doses may be required.

- Although previously recommended, doubling the ICS dose has since been shown to be ineffective in reducing the need for oral corticosteroids or avoiding unscheduled urgent medical visits.
- During a mild exacerbation detected by a decline in PEF, even a single high dose given in addition to doubling the ICS dose is more effective in increasing the rate of recovery than doubling the dose alone.

Small regular doses of ICS are markedly effective in preventing exacerbations. Meta-analyses of many studies of varying lengths (4 weeks to > 1 year) have shown the number needed to treat (NNT) to prevent one major exacerbation is 3, based on a daily dose of FP 100 mcg or equivalent and 2 based on a dose of 500 mcg.

**Table 1. ICS dose equivalents: what is meant by low, medium and high daily doses?**

<table>
<thead>
<tr>
<th>Daily ICS dose</th>
<th>CIC*</th>
<th>BDP–HFA**</th>
<th>FP**</th>
<th>BUD**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>80–160 mcg</td>
<td>100–200 mcg</td>
<td>100–200 mcg</td>
<td>200–400 mcg</td>
</tr>
<tr>
<td>Medium</td>
<td>160–320 mcg</td>
<td>200–400 mcg</td>
<td>200–400 mcg</td>
<td>400–800 mcg</td>
</tr>
<tr>
<td>High</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 800 mcg</td>
</tr>
</tbody>
</table>

ICS: inhaled corticosteroid; LABA: long-acting beta₂ agonist; CIC: ciclesonide; BDP–HFA: beclomethasone dipropionate; FP: fluticasone propionate; BUD: budesonide

*ex actuator dose

**ex valve dose

**Dosing considerations for combination LABA–ICS therapy**

**Budesonide plus eformoterol (Symbicort)**

In patients taking budesonide plus eformoterol (Symbicort) it may be reasonable to add extra doses in response to worsening asthma control (maintenance and reliever regimen). This practice may reduce the risk of moderate-to-severe exacerbations over the medium term (compared with fixed dosing plus as-needed SABA), but it is not known whether these benefits are due to improved long-term control (secondary prevention of exacerbations) or to very early treatment of incipient exacerbations (primary prevention of severe exacerbations), or to both.

**Fluticasone plus salmeterol (Seretide)**

- When patients taking Seretide experience worsening asthma control, it may be reasonable to administer high-dose fluticasone in addition to maintenance (e.g. via a separate inhaler). However, the efficacy of this approach has not been assessed in randomised clinical trials. In all clinical trials of Seretide, exacerbations were managed with increased SABA use and/or oral corticosteroids.
- Based on limited data, doubling the dose of Seretide (and therefore doubling the dose of salmeterol) for 2 weeks in patients with stable asthma appears to be well tolerated.

Oral corticosteroids should be given within 24–48 hours when high-dose ICS or an increase in ICS–LABA combination treatment fails to control an exacerbation.

**When is an exacerbation an emergency?**

Urgent treatment is needed if any of the following apply in adults:

- The patient is experiencing severe respiratory distress.
- Response to reliever is not immediate and sustained for at least 3 hours.
- There is further worsening of symptoms despite treatment.
- The patient’s asthma action plan indicates the need for urgent medical care.
- Peak expiratory flow remains less than 60% predicted or personal best after reliever medication taken as instructed.
Managing asthma exacerbations in children

Practice points

- A short (up to 5 days) course of oral corticosteroids (prednisolone 1 mg/kg up to 60 mg daily) is the current standard treatment for severe exacerbations. Closely monitor response to treatment. (I)
- Children who are taking regular preventive medication should continue taking the same dose during an exacerbation. (II)
- When administering a SABA via MDI during an exacerbation, use a spacer. (III-1)
- Merely doubling the maintenance ICS dose is not effective in managing exacerbations in children. (II)

In children, the majority of acute exacerbations are triggered by common respiratory viral infections. These do not respond to increased ICS doses.18 Children who are taking regular preventive medication should continue taking the same dose.

Provide children and their parents with a comprehensive asthma action plan for recognising and managing acute episodes. See Asthma action plans.

Oral corticosteroids

Severe exacerbations should be managed with oral corticosteroids.
- Oral corticosteroids are indicated in asthma attacks sufficiently severe to warrant presentation to an emergency department.10
- Parent- or carer-initiated oral prednisolone early in an acute asthma episode should be reserved for children with a history of severe episodes likely to result in hospital admission. This practice has not been shown to benefit children with mild viral wheezing.28

Short-acting beta₂ agonists

There is strong evidence that inhaled short-acting beta₂ agonists (SABAs) are effective in the management of exacerbations.
- During exacerbations, SABAs should be administered via metered dose inhaler (MDI) and spacer.1,13
- Delivery of SABA via MDI and spacer is equally effective as nebulisation in children with moderate-to-severe acute asthma.13
- Doses are as for acute asthma episodes:
  - For children under 6 years old, 4–6 puffs salbutamol 100 mcg/dose (or equivalent) via MDI, repeated after 20 minutes or as necessary until control is achieved.
  - For children 6 years and over, 8–12 puffs salbutamol 100 mcg/dose (or equivalent) via MDI, repeated after 20 minutes or as necessary until control is achieved.

For more information on SABA doses and administration of SABAs during an exacerbation, see Acute asthma.

Inhaled corticosteroids

- Doubling the dose of ICS at the onset of an exacerbation (indicated by a decrease in PEF, nocturnal cough or wheeze or increased SABA requirement) is ineffective in improving lung function and controlling symptoms.18
- In children with acute exacerbations insufficiently severe to necessitate hospitalisation, short-term use of high-dose ICS (equivalent to 1600 mcg budesonide daily) in addition to SABA might be an effective alternative to oral corticosteroids.29
- Oral corticosteroids are more effective than ICS in children with severe acute asthma.30,31

Leukotriene receptor antagonists

In children who have intermittent asthma induced by viral upper respiratory tract infection, a short course of oral montelukast (7–10 days) commenced at the onset of infection may reduce the severity of that episode.32

See Asthma action plans.
Complementary and alternative medicine in asthma

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ask about use of complementary medicines and advise patients about potential hazards.</td>
<td>✓</td>
</tr>
<tr>
<td>Warn patients that echinacea and royal jelly can precipitate life-threatening anaphylaxis in predisposed individuals with asthma.</td>
<td>IV</td>
</tr>
<tr>
<td>Encourage a good therapeutic partnership with the patient by approaching the issue in an open, sympathetic manner and avoiding a rigid dismissal of complementary therapy.</td>
<td>✓</td>
</tr>
<tr>
<td>Encourage patients to choose TGA-assessed products in preference to unregulated products and make sure they understand that the Aust L code does not indicate that the product is effective.</td>
<td>✓</td>
</tr>
<tr>
<td>Monitor effects of complementary therapies as for conventional therapies.</td>
<td>✓</td>
</tr>
<tr>
<td>There is evidence for subjective improvements in asthma symptoms with some breathing techniques, but not for improvement in lung function.</td>
<td>I</td>
</tr>
<tr>
<td>Current evidence does not support the use of manual therapies (chiropractic, osteopathy and related modalities) for patients with asthma.</td>
<td>I</td>
</tr>
<tr>
<td>Current evidence does not support the use of acupuncture as a treatment for asthma.</td>
<td>I</td>
</tr>
<tr>
<td>Physical training improves cardiopulmonary fitness but does not improve lung function or symptoms.</td>
<td>I</td>
</tr>
<tr>
<td>Based on current evidence, health professionals can advise patients that homeopathy is ineffective.</td>
<td>I</td>
</tr>
<tr>
<td>Although some traditional Chinese medicines might be beneficial in asthma, they cannot yet be recommended for clinical use based on current efficacy and safety data.</td>
<td>I</td>
</tr>
<tr>
<td>Alternative diagnostic tests should not be used in the diagnosis of asthma and allergies.</td>
<td>✓</td>
</tr>
</tbody>
</table>

Note: various definitions of ‘complementary’ and ‘alternative’ medicine are used in Australia and overseas. Here CAM refers to the wide range of medical and health care systems, practices and products that are not currently considered to be part of conventional medicine.1

Since this is a large and growing field, an exhaustive review of evidence is beyond the scope of this handbook.

For more information on CAM and asthma, see the information resources at the end of this chapter.

Health professionals can increase patients’ understanding of expected health effects of complementary medicine through a non-judgemental approach and an honest discussion of what is known about their effects and by avoiding a rigid dismissal of complementary therapy. When treating a patient with asthma:

- always ask about use of complementary products when taking a medication history
- consider potential effects on asthma and interactions between complementary medicines and prescribed conventional asthma medications
- approach the issue of CAM in an open, sympathetic manner, acknowledging the patient’s experience with complementary therapies and asthma control
- be aware of potential hazards of CAM and advise patients about these (Table 1).

If introducing complementary therapies, any dose reduction in conventional therapy should be conducted under the supervision of a medical practitioner to avoid adverse outcomes.
Complementary and alternative medicine

Clinical evaluation and regulation of CAM

Overall, there is less documented evidence for the effectiveness and safety of complementary therapies than for conventional therapies.

- The majority of published studies have examined the complementary therapy as adjunctive therapies given in addition to patients' usual medical care (usually inhaled corticosteroids).
- Clinical trials of complementary therapies have not always collected safety data (e.g. frequency of exacerbations, deaths), as is routinely done in registration trials of conventional drug therapies.
- Some studies have not recorded lung function outcomes or measures of airway inflammation.

The placebo effect might explain the findings of those trials that have reported improvements in symptoms or reduction in reliever use, but failed to demonstrate lung function improvements. Regardless of the mechanism for observed effects, such a modality may confer clinically significant benefits (e.g. reduction in adrenergic adverse effects resulting from reduced reliever use, reduced response to allergens, reduced mast cell tachyphylaxis).3–5

In Australia, most complementary medicines are regulated by the Therapeutic Goods Administration to ensure that they conform with lists of permitted ingredients, and are manufactured under the same standards as pharmaceuticals.6 Products that have undergone this assessment are indicated on the label by the codes Aust L or Aust R. Most complementary medicines are Aust L coded. Patients should be encouraged to choose TGA-assessed products in preference to unregulated products (e.g. those obtained via the internet), and advised that the Aust L code does not indicate that the product is effective.

Most complementary therapists are not required to be registered with a professional association regulating their practice, so the ‘buyer beware’ principle applies.

Table 1. Some potential adverse effects of CAM

<table>
<thead>
<tr>
<th>CAM modalities</th>
<th>Potential safety issues</th>
</tr>
</thead>
</table>
| Any product                    | Interactions between complementary and conventional medicines
|                                 | Refer to Australian Drug Reactions Advisory Committee (ADRAC) reports for information about specific products (www.tga.gov.au/adr/adrac.htm). |
| Echinacea                      | Allergic reactions8–10                          |
| Royal jelly                    |                                                |
| Willow tree bark extracts (aspirin) |                                              |
| Camomile                       |                                                |
| Traditional Chinese medicines  | • Mahuang (ephedra) preparations contain ephedrine, associated with headache, nausea, irritability, restlessness, insomnia, tachycardia, hypertension, motor disturbances.11
|                                | • Heavy metal contamination12                   |
|                                | • Adulteration with conventional pharmaceuticals, e.g. nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids.12–14 |
| Indian Ayurvedic medicines     | • Heavy metal contamination14,15               |
|                                | • Adulteration with conventional pharmaceuticals, e.g. NSAIDs, corticosteroids14 |
| Ginkgo biloba                  | • Headache, nausea, dizziness, palpitations, allergic skin reactions, bleeding (rarely).8
|                                | • Interaction with warfarin.9                  |
| Vitamin and mineral supplements| • Toxic in high doses 7 Refer to Approved Product Information |

Breathing techniques

There is evidence for subjective improvements in asthma symptoms with some breathing techniques, but not for improvement in lung function. Studies should be interpreted with reference to the baseline prevalence of dysfunctional breathing among patients with asthma.16

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Complementary and alternative medicine

Buteyko breathing technique
The Buteyko breathing technique is a system of breathing exercises that focuses on breathing through the nose, hypoventilating and avoiding deep breaths. It is based on the theory that slowing the rate of breathing will raise levels of carbon dioxide, a natural bronchodilator, and will therefore result in bronchodilatation and symptomatic improvement. Controlled studies of the Buteyko breathing technique have demonstrated symptomatic improvement and reduction in the use of reliever medication in some patients,17–19 but have not demonstrated changes in carbon dioxide levels, lung function measures or measures of airway inflammation.

Yoga
Some studies of yoga have reported subjective improvement of asthma symptoms but not examined lung function or airway inflammation. Overall, the evidence for benefit in patients with asthma is inconclusive.20–23

Dietary modification
While patients with demonstrated food allergies should be advised to avoid food allergens, routine dietary restrictions in patients with asthma are not beneficial.

Food supplements
• Based on very limited data from randomised clinical trials, food supplements that might offer some benefit as adjunctive treatment in the control of asthma symptoms include omega-3 fatty acids (fish oils),24-26 lycopene27 and selenium supplements.28 Except for omega-3 fatty acids, none have been shown to improve objective lung function parameters.
• Other food supplements that have been assessed in the management of asthma but cannot be recommended based on current evidence include Lactobacillus acidophilus,29 vitamin C,30 vitamin E and magnesium supplements.31–33
For information on dietary approaches to asthma therapy, see Diet and asthma.

Manual therapies
Current evidence does not support the use of manual therapies (chiropractic, osteopathy and related modalities) for patients with asthma. (I)
• Chiropractic manipulation is not effective in improving asthma symptoms, lung function, or reducing medication requirement in adults and in children with persistent asthma.35–37
• Controlled studies of osteopathy and cranial therapy in patients with asthma or allergic rhinitis have shown inconsistent results for effects on lung function and no convincing evidence of effectiveness in reducing medication requirements or symptoms.38,39
• Massage therapy has been reported to reduce reliever medication requirement in children.40

Acupuncture
Current evidence does not support the use of acupuncture as a treatment for asthma.41,42

Exercise therapies
Various programs of physical training have been assessed in adults and children.
• Physical training improves cardiopulmonary fitness but does not improve lung function or symptoms.43
• Swimming has not been shown to improve lung function or reduce medication requirement in limited available clinical trial evidence.44

Tai chi and qigong
The effects of tai chi and qigong on asthma have not been investigated in randomised controlled trials.

Medicinal therapies

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Homoeopathy
Based on current evidence, health professionals can advise patients that homoeopathy is ineffective.45–47

Herbal remedies and traditional Chinese medicine
Many traditional Chinese medicines are the subject of ongoing study, and some have shown promising results in recent studies. However, effectiveness and safety is yet to be defined and their use is not currently recommended. Some have been associated with adverse effects (Table 1).

Psychological therapies
The use of psychological interventions in adults or children with asthma is not well supported.48,49
• Limited data suggest heart rate-variability biofeedback may help reduce asthma medication requirement.50
• There is no convincing evidence for effectiveness of relaxation therapies in the management of asthma.51
• Hypnotherapy may be an effective adjunct to asthma treatment in some patients, based on limited data.52

For information on links between asthma and mental illness, see Other comorbidities.

Other therapies
• There is no published evidence from randomised controlled trials for the effects of aromatherapy, vega matrix regeneration therapy or radionics (psionic medicine, dowsing) in the treatment of asthma.
• Clinical trials assessing reflexology in patients with asthma have not demonstrated improvements in lung function, symptoms or medication requirement, compared with standard care or sham treatment.53,54
• Current evidence does not support the use of enzyme-potentiated immunotherapy, which involves adding allergen extracts to a suspension of freshly drawn patient blood, mixing with beta glucuronidase and injecting the resultant solution intradermally.

For information on immunomodulatory therapy, see Asthma and Allergy .

Alternative diagnostic tests
Practice point
Alternative diagnostic tests should not be used in the diagnosis of asthma and allergies. ✓

Key points
• There is no strong evidence for a beneficial role of CAM in asthma therapy.42
• There is some evidence that the Buteyko breathing technique may reduce asthma symptoms and requirement for reliever medication, but there is not evidence for an improvement in lung function or airway inflammation.
• There is preliminary evidence that some Chinese herbal medications may benefit some patients with asthma, but their effectiveness and safety have not yet been established in clinical trials. Their use is not recommended in clinical practice.
• A range of other CAM modalities currently used for asthma have not been proven effective, and some carry potential adverse effects.

Information resources
For patients

For health professionals

Various unorthodox tests used by alternative therapy practitioners have not been shown to be reliable or reproducible when subjected to formal study. The following tests are not appropriate for the diagnosis of asthma and allergies or to guide asthma management:55
• cytotoxic food testing (Bryan’s test)
• oral provocation and neutralisation
• Vega testing (electrodermal testing)
• kinesiology
• radionics (psionic medicine, dowsing)
• iridology
• pulse testing
• hair analysis
• tests for ‘dysbiosis’
• blood assays for essential fatty acids, vitamins and minerals.
Diet and asthma

People with asthma are often interested in the effect that diet may have on their symptoms. While food allergens are uncommon triggers for asthma, health professionals need to be aware of the issues and myths surrounding diet and asthma: many patients with chronic diseases seek alternative advice from other practitioners, including those with little experience and often no scientific basis for their recommendations. Patients should be particularly wary of any advice that claims that a change in diet can cure asthma and eliminate the need for medication.

Food as a trigger for asthma

Food allergy

- Immunoglobulin (IgE)-mediated reaction to a food protein in a patient with a food protein allergy can result in bronchoconstriction.
- The most common foods that may cause IgE-mediated food allergy are peanuts, tree nuts, fish, shellfish, milk, eggs, wheat and soy.
- Most children grow out of their allergy to milk, eggs, wheat and soy, but allergies to peanuts, tree nuts, fish and shellfish can to be lifelong.1
- The only dietary treatment currently available for proven food allergy is complete avoidance of the food protein.
- The presence of asthma is a risk factor for serious food allergic reactions.
- Food allergy must be confirmed by appropriate tests done by an allergy/immunology specialist.

For more detailed information on food allergy and asthma, go to Asthma and allergy.

Food chemical intolerance

- Some food chemicals have been reported to trigger symptoms of asthma.
- There is currently no evidence that avoidance of food additives has any routine role in asthma management.
- Investigation for food chemical intolerance must be conducted in a specialised unit and involves investigation with double-blind, placebo-controlled challenges.

<table>
<thead>
<tr>
<th>Food Trigger</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphur dioxides</td>
<td>Considered the most reactive food additive;</td>
</tr>
<tr>
<td></td>
<td>Ingestion or inhalation can cause bronchoconstriction; may affect up to 5–10% of people with asthma.2,3</td>
</tr>
<tr>
<td>Monosodium glutamate (MSG)</td>
<td>The existence of MSG-induced asthma has not been firmly established.4</td>
</tr>
<tr>
<td>Tartrazine</td>
<td>No firm conclusions regarding the effects of tartrazine exclusion in asthma.5</td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>There is no evidence to recommend that people with aspirin sensitivity should avoid naturally occurring dietary salicylates.</td>
</tr>
</tbody>
</table>

SUMMARY OF PRACTICE POINTS

| Food allergens are uncommon triggers for asthma in any age group; diet should not be restricted unnecessarily. | II |
| Treatment of proven food allergies involves avoidance of foods known to cause symptoms. | ✔ |
| Skin prick tests or RAST will be positive for foods that cause IgE-mediated food allergies. | ✔ |
| There is no medical foundation for the widely held view that dairy products increase mucus secretions. | ✔ |
| Weight reduction in overweight or obese people with asthma may help to reduce asthma symptoms. | III |
| Exclusive breastfeeding for the first 6 months of life should be encouraged for all infants. Breastfeeding can protect against allergic rhinitis, wheezing, asthma and atopy in children. | III |

Foot chemical intolerance

- Some food chemicals have been reported to trigger symptoms of asthma.
- There is currently no evidence that avoidance of food additives has any routine role in asthma management.
- Investigation for food chemical intolerance must be conducted in a specialised unit and involves investigation with double-blind, placebo-controlled challenges.

<table>
<thead>
<tr>
<th>Food Trigger</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphur dioxides</td>
<td>Considered the most reactive food additive;</td>
</tr>
<tr>
<td></td>
<td>Ingestion or inhalation can cause bronchoconstriction; may affect up to 5–10% of people with asthma.2,3</td>
</tr>
<tr>
<td>Monosodium glutamate (MSG)</td>
<td>The existence of MSG-induced asthma has not been firmly established.4</td>
</tr>
<tr>
<td>Tartrazine</td>
<td>No firm conclusions regarding the effects of tartrazine exclusion in asthma.5</td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>There is no evidence to recommend that people with aspirin sensitivity should avoid naturally occurring dietary salicylates.</td>
</tr>
</tbody>
</table>
Diet and asthma

Food as a potential treatment for asthma

Studies are ongoing into the potential role of dietary supplements for the treatment of asthma. There is currently no conclusive evidence for using dietary supplements, including probiotics or antioxidants, in the treatment of asthma.

Weight loss in the obese patient

Epidemiological studies have shown an association between increasing body mass index (BMI) and increased prevalence of diagnosed or reported asthma in both adults and children.12–15 Weight reduction in both obese and morbidly obese asthmatics has been associated with improved symptom scores and reduced medication requirements.16,17 Health professionals should consider discussing weight loss in overweight and obese patients with asthma.

Infant feeding

Exclusive breastfeeding for the first 6 months of life should be encouraged for all infants. In the general population and in atopic families, breastfeeding can protect against allergic rhinitis, wheezing, asthma and atopy in children.18 If exclusive breastfeeding is not possible, supplementation with cows’ milk formula is recommended. There is no evidence to support a soy based formula over a cows’ milk formula in infants at risk of developing asthma.19 A hydrolysed formula may be indicated for infants at high risk of developing asthma.20,21

Food Treatment | Evidence
--- | ---
Omega-3 polyunsaturated fatty acids | Little evidence to recommend that people with asthma supplement or modify their dietary intake of marine omega-3 fatty acids in order to improve their asthma.6
Antioxidant vitamins C and E | Evidence insufficient to recommend a specific role for vitamins C or E in the treatment of asthma.7,8
Magnesium | Meta-analysis found that magnesium supplementation may be beneficial in children with mild-to-moderate asthma; further research required before it can be recommended.9
Selenium | Currently insufficient evidence that selenium supplementation is of benefit in asthma.10
Sodium | Currently insufficient evidence that a low sodium diet will be of benefit in asthma.11

Practice points

• Food allergens are uncommon triggers for asthma in any age group; diet should not be restricted unnecessarily. (II)
• Treatment of proven food allergies involves avoidance of foods known to cause symptoms. ✓
• Skin prick tests or RAST will be positive for foods that cause IgE-mediated food allergies. ✓
• There is no medical foundation for the widely held view that dairy products increase mucus secretions. ✓

Practice tips

• People with asthma should follow a diet appropriate for general good health, including consumption of fresh fruits and vegetables, lean meats and fish, wholegrain breads and cereals, dairy products and oils low in omega-6 fatty acids such as olive oil or canola oil.
• People with asthma on high-dose corticosteroids should use calcium supplementation.
• When food choices are restricted, the advice of a dietitian should be sought to ensure that the dietary intake continues to meet nutrient and energy needs.

Practice points

• Exclusive breastfeeding for the first 6 months of life should be encouraged for all infants. (III)
• Breastfeeding can protect against allergic rhinitis, wheezing, asthma and atopy in children. (V)
Asthma and allergy

SUMMARY OF PRACTICE POINTS | LEVEL OF EVIDENCE
---|---
Inhalant allergens are a major trigger for asthma and wheezing in allergic individuals. In sensitised individuals, exposure to house dust mite, pollen, domestic pets, moulds or cockroaches can trigger asthma attacks or worsen symptoms. | II
There is no definitive evidence that strategies to reduce exposure to house dust mites are effective in controlling asthma. However, some patients may benefit from these strategies. | ✓
Food allergens are uncommon triggers for asthma. | II
Acute food allergen-induced respiratory symptoms may be due to anaphylaxis, not asthma – particularly if associated with urticaria or angioedema. | ✓
Ensure responsible food allergens are accurately identified and appropriate avoidance strategies are instituted. This will usually entail referral to a specialist with experience in allergy and clinical immunology. | ✓
Intranasal corticosteroids are the most effective medications for controlling symptoms of allergic rhinitis. | I
Specific allergen immunotherapy might reduce the risk of childhood rhinitis progressing to asthma. | II
Specific immunotherapy (SIT) has clear therapeutic benefits in asthma | I
Pre-treatment with less-sedating H1-antihistamines can significantly reduce local and systemic adverse reactions to immunotherapy. | II
Immunotherapy is contraindicated in patients with severe or unstable asthma. | IV

There is a strong association between allergy (sensitisation to allergens) and asthma, although these processes appear to develop independently.¹

- Asthma is regarded as one of the final stages of the ‘atopic march’, which frequently begins in infancy as food allergy and atopic dermatitis. As many as 80% of infants with early evidence of allergic disease will go on to develop asthma or allergic rhinitis.²
- Early allergic sensitisation is a major risk factor for persistent wheezing and airway hyperreactivity.³ ⁴
- Allergic individuals are over three times more likely to develop asthma⁵ and airway hyperresponsiveness.⁶ Around 70–90% of individuals with established asthma show hypersensitivity to one or more allergens,⁶ ⁷ and this proportion is higher among children than adults.

**The concept of one airway**

There is growing awareness of close functional relationships between the upper and lower respiratory tract.⁸ ⁹

- Patients with allergic rhinitis frequently have associated bronchial hyperreactivity.¹⁰
- An estimated 60–80% of asthma patients have coexisting allergic rhinitis¹¹ or inflammation of paranasal sinuses.¹²
- Treatment of allergic rhinitis improves asthma control.¹³

---

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Inhalant allergens as triggers for asthma

Inhalant allergens are major triggers for asthma and wheezing in allergic individuals.

- In sensitised individuals, exposure to house dust mite, pollen, domestic pets, moulds and cockroaches have been shown to trigger asthma attacks or worsen symptoms.\(^{14-20}\) However, allergen exposure does not necessarily correlate with clinical symptoms, because patients may be sensitised to multiple allergens and have other triggers (e.g. viral infections).
- Rarely, asthma may be triggered by inhalation of a food allergen, e.g. when cooking seafood.

Strategies to reduce exposure to inhalant allergens

Various allergen avoidance measures have been attempted. Although some can reduce allergen exposure, many are ineffective and without clear evidence that they are useful in controlling asthma.\(^{21}\) Because many allergen avoidance measures can entail significant expense and inconvenience, it is important that patients are aware that these have not been proven to be effective in controlling disease.

- **House dust mite** – exposure to house dust mite increases the risk of asthma exacerbations in sensitised individuals with asthma.\(^{22}\) Reduction in exposure to allergen triggers such as house dust mite is possible,\(^{23-26}\) but may be difficult to achieve in humid climates. Meta-analyses have found no conclusive evidence that house dust mite reduction strategies are effective as prophylactic treatment in established asthma.\(^{21,27}\)

- **Pollens** – Sensitisation to pollen from grasses, weeds and trees is common in people with asthma. Asthma symptoms can worsen during periods of high pollen counts, such as seasonal changes and after thunderstorms.\(^{33}\) Pollens can be carried in the air for miles, making it difficult to effectively avoid exposure. There is limited evidence for the effectiveness of pollen avoidance strategies or their role in controlling asthma or other allergic disease.

- **Moulds** – exposure to aerosol moulds occurs both indoors and outdoors. Indoor exposure has been associated with wheezing and peak expiratory flow variability.\(^{34,35}\) Sensitisation to moulds (\textit{Alternaria} species) has been reported to be a strong risk factor for asthma in arid climates. Although some strategies may reduce indoor mould levels, a role in improving asthma control has not been demonstrated.

Although other indoor allergens such as cockroaches may be relevant in Australia, there is no evidence that strategies to reduce exposure to these allergens are effective or improve asthma control.

Detailed information on allergen avoidance measures for patients is available from the Australasian Society of Clinical Immunology and Allergy (www.allergy.org.au).

Food allergy and asthma

Food allergens are uncommon triggers for asthma in any age group: as few as 2.5% of people with asthma react to foods in blinded challenges.36

Sensitisation to foods is a common early manifestation of allergic disease, and a significant proportion of children with food allergies will develop inhalant allergies and allergic airway disease (asthma and/or allergic rhinitis).37–39

- The incidence of food allergies in children appears to be increasing.40,41 Food allergies to egg, milk and soy usually resolve in the preschool years, although allergies to other foods e.g. peanuts, nuts and shellfish are more likely to persist.
- In a minority of sensitised individuals, exposure to foods can trigger anaphylaxis with associated wheezing, which should be distinguished from asthma. Patients with anaphylaxis require treatment with adrenaline as a priority over other treatments such as bronchodilators.
- The presence of asthma has been shown to be a risk factor for fatal and near-fatal food-induced anaphylactic reactions.42,43

All people with food allergies should be referred to a specialist with specific expertise in allergy/immunology for assessment for relevant allergens, and for treatment advice, including food avoidance and adrenaline prescription where appropriate. Rarely, food allergens can trigger asthma when inhaled.

Poorly controlled asthma is a risk factor for fatal reactions in individuals with a history of anaphylactic allergic reactions.36,42

Allergy tests

Correct identification of allergen trigger factors offers opportunities for appropriate allergen avoidance or for disease modification by immunotherapy, which may minimise the need for long-term drug therapy. Allergy testing:

- is recommended for patients with persistent asthma or who require regular preventer therapy.
- may be considered in patients with asthma and allergic rhinitis to clarify whether allergens are contributing to disease. If allergy is not present there is no need to consider anti-allergy measures.
- should be considered for those who request it.

Currently available allergy tests detect the presence of allergen-specific IgE. The presence of these antibodies indicates sensitisation but does not necessarily predict the presence, pattern or severity of clinical reactivity. Allergy tests should be interpreted in the clinical context in consultation with an allergy specialist or a medical specialist trained in allergy. In Australia, testing with ryegrass and house dust mite will detect more than 95% of the IgE reactors in the community.

- Allergy skin prick tests (SPT) detect the presence of allergen-specific IgE bound to mast cells in the skin and reflect systemic sensitisation. These in vivo tests must be performed by experienced, trained staff using standardised techniques. The tests may be inhibited by antihistamines and some other medications (e.g. tricyclic antidepressants). The positive control allows correct interpretation of validity of the allergy tests.
- Radioallergosorbent tests (RAST) or related assays detect the presence of circulating allergen-specific IgE. These tests are not affected by antihistamines.
and other medications, but are less sensitive than SPT. These tests are also more expensive.

- Some so-called ‘allergy tests’, including Vega tests, bioelectric tests, pulse tests and applied kinesiology, have no scientific basis and therefore have no place in the clinical assessment of asthma.

### Allergic rhinitis and asthma

Allergic rhinitis and asthma frequently co-exist and allergic rhinitis is a recognised risk factor for developing asthma.44-46

For more information on allergic rhinitis and asthma, see Allergic rhinitis and the patient with asthma. A guide for health professionals (available at www.nationalasthma.org.au).

### Diagnosis

Diagnosis of allergic rhinitis requires:

- detailed and accurate history (family and personal history of allergic conditions, environment, occupation)
- physical examination (focus on the nose, throat, eyes and ears, spirometry if indicated)
- allergy investigation.

Classical symptoms of itching, nasal irritation, watery rhinorrhoea and eye symptoms favour a diagnosis of allergic rhinitis, but they are not specific and may be caused by a viral infection or by vasomotor rhinitis. The absence of classical symptoms does not exclude the diagnosis of allergic rhinitis. Blockage is often the predominant symptom in persistent disease.

### Differential diagnosis

- Vasomotor rhinitis – may present with clear watery nasal drainage following changes in temperature.
- Bacterial infection – may present with purulent nasal drainage.
- Nasal polyps – may present with congestion and loss of sense of smell.
- Recurrent viral upper respiratory infections.

### Drug therapy

A variety of over-the-counter treatments are available for allergic rhinitis and the majority of Australian adults now self-medicate their condition.

#### Inhaled nasal corticosteroids (INCS)

If continuous treatment is required, an INCS is the first-choice treatment in adults and children, especially in patients with asthma.46–49

- There is no clear evidence that one preparation is more clinically effective than another, although once-daily dosing regimens may improve adherence.
- Mometasone furoate nasal spray can be used in children over 3 years old; budesonide nasal spray can be used in children over 6 years old.

#### Antihistamines

Oral H1-antihistamines are useful in patients with milder allergic rhinitis, particularly in children who do not tolerate INCS.

- Antihistamines are less effective than INCS, particularly in controlling congestion.
- Second-generation, less sedating antihistamines should be used in preference to more sedating antihistamines.
- Less-sedating oral antihistamines are appropriate first-line therapy in children and adults with mild allergic rhinitis or intermittent allergic rhinitis.
- Intranasal antihistamines can be used as alternatives to oral antihistamines.49

#### Other agents

- In patients with asthma, leukotriene antagonists may also contribute to control of allergic rhinitis symptoms.50
- Intranasal decongestants have a limited role in the management of allergic rhinitis because they should only be used for very short courses (up to 5 days’ maximum). Repeated or long-term use can cause rebound swelling of nasal mucosa necessitating dose...
escalation (rhinitis medicamentosa), with a risk of atrophic rhinitis. Intranasal decongestants might be considered in a patient with severe nasal congestion where rapid onset of action required until the full effect of INCS is achieved.

- Oral decongestants are indicated for short-term (e.g. 2–3 days) use only. Rebound congestion (rhinitis medicamentosa) and insomnia can occur with more prolonged use. They are contraindicated in patients with hypertension or coronary artery disease, and should be used with caution in people aged over 65 years, in patients with benign prostatic hyperplasia and those taking multiple medications.
- Anticholinergic sprays (e.g. ipratropium bromide) are effective in managing persistent rhinorrhea.46
- Ocular anti-allergy preparations (antihistamines, decongestants, mast-cell stabilising agents) may be considered if allergic conjunctivitis persists despite INCS. Check contraindications (e.g. glaucoma, pregnancy) for specific agents.
- Oral corticosteroids should be avoided as a treatment for allergic rhinitis. In exceptional circumstances, their use might be considered in consultation with an allergy specialist. There is no role for intramuscular corticosteroid therapy as a treatment for rhinitis due to the risk of local muscle necrosis and hypothalamic-pituitary suppression.

### Immunotherapy (desensitisation)

Immunotherapy should be considered for patients with moderate-to-severe allergic rhinitis who have not responded to optimal medical therapy and allergen avoidance. See Immunomodulatory therapy.

### Specialist referral for patients with allergic rhinitis

Referral to an allergy specialist or medical specialist trained in allergy is recommended if the patient:
- has poorly controlled asthma or severe allergic comorbidities such as eczema and food allergies
- has complications such as resistant obstruction, anosmia, sinus disease, ear problems, persistent purulent drainage, behavioural effects
- has persistent and/or unresponsive symptoms
- has associated food allergy
- requests referral.

Referral to an ear, nose and throat specialist is recommended if the patient:
- has constant unilateral obstruction
- has a polyp that is unresponsive to initial INCS therapy
- has complications such as resistant obstruction, anosmia, sinus disease, ear problems, persistent purulent discharge.

### Immunomodulatory therapy

There is good evidence that immunomodulatory therapy can modify allergic immune responses and improve symptoms in established asthma. There is also accumulating evidence that ultimate lung function is ‘set’ in the first years of life, thus, there is increasing need to develop early interventions to prevent chronic airway inflammation and remodelling in early life.51,52

Objectives of immunomodulation are:
- to induce long-lasting changes in immune responses and clinical improvement in existing asthma and allergic disease
- to prevent allergic disease progression in children by reducing the risk of new sensitisations and the development of asthma.

Currently available immunomodulatory strategies include:
- conventional (subcutaneous) specific immunotherapy (SIT)
- sublingual immunotherapy (SLIT)
- Anti-IgE monoclonal antibody therapy.

More experimental techniques with future potential include:

### Practice points

- Intranasal corticosteroids are the most effective medications for controlling symptoms of allergic rhinitis. (I)
- Specific allergen immunotherapy may reduce the risk of childhood rhinitis progressing to asthma. (I)

**Pharmacy practice tip**

Refer patients to their GP for assessment if any of the following apply:
- Symptoms are not controlled despite the use of over-the-counter INCS and antihistamine
- Symptom control requires long-term (>1 month) treatment
- The person has other medical conditions
- Quality of life is seriously affected
- Rhinitis is associated with pain, loss of sense of smell or loss of hearing.
• Allergen-peptide immunotherapy
• Monoclonal antibodies to molecular targets (e.g. cytokines).

Conventional (subcutaneous) specific immunotherapy (SIT) in asthma

Specific allergen immunotherapy involves the administration of allergen extracts to induce persistent clinical tolerance in patients with allergen-induced symptoms. This approach currently offers the only ‘curative’ therapy for allergic propensity.

• SIT is currently the most common form of immunotherapy used in Australia.

• Beneficial effects of SIT include improvements in symptom scores, reduction in medication usage and reduced bronchial hyper-responsiveness with specific immunotherapy for house dust mite or other inhalant allergens (including pollens, moulds or animal dander).

• Clinical benefits are associated with long-lasting immunological changes, including increased regulatory T cell activity and suppression of allergic (T helper type 2) immune responses. This is associated with switching of allergen-specific B cells towards IgG4 production.

• Pre-treatment with H1-antihistamines can significantly reduce local and systemic adverse reactions to immunotherapy.

• In most centres, SIT for inhalant allergens is continued for 3 years if there is a clinical response, although practice varies.

Indications for SIT

Referral for immunotherapy assessment should be considered for patients with asthma with any of the following who are likely to comply with the immunotherapy regimen:

• Suspected allergen triggered disease, where allergen avoidance has been ineffective or not possible
• Mild-to-moderate stable asthma (FEV₁ > 70% predicted), where there is a clear demonstration of a specific allergen trigger (symptoms on exposure plus allergen-specific IgE)
• Co-existing allergic rhinitis
• Request for immunotherapy.

Immunotherapy is contraindicated in patients with severe or unstable asthma and those using beta blockers.

Only practitioners with training and experience in the management of both asthma and immunotherapy should make the decision to start immunotherapy. It is common for GPs to administer maintenance immunotherapy under specialist guidance; this should be done according to the relevant prescribed procedure and with the recommended precautions.

Immunotherapy practitioners must be skilled in resuscitation and have the appropriate facilities immediately available, including the following:

• Adrenaline 1:1000 for intramuscular use (adrenaline is the drug of choice for the immediate management of systemic reactions to immunotherapy)
• Oxygen
• An inflatable bag and mask ventilator
• A nebuliser and bronchodilator nebuliser solution or a bronchodilator metered dose inhaler with spacer device
• Needles and tubing for intravenous access
• Intravenous fluids suitable for volume replacement
• Parenteral antihistamine
• Parenteral corticosteroid

A second appropriately trained health care professional should be present during immunotherapy to assist if resuscitation is required.

Practice points

• Specific immunotherapy (SIT) has clear therapeutic benefits in asthma. (I)
• Pre-treatment with less-sedating H1 antihistamines can significantly reduce local and systemic adverse reactions to immunotherapy. (II)
• Immunotherapy is contraindicated in patients with severe or unstable asthma. (IV)

Adverse effects

SIT has been under-utilised as a therapeutic option because of potential side effects (including anaphylaxis), although these can be minimised when it is performed appropriately.

• Local reactions (mild swelling and erythema at the site of the injection) occur with most injections and typically persist for 24 hours without serious effects.
• Systemic reactions include sneezing, bronchospasm, urticaria and anaphylaxis with hypotension and collapse. When these occur, they are usually evident within 30 minutes of injection.

Asthma should be monitored prior to, during, and after immunotherapy.
• Spirometry or peak flow monitoring must be performed prior to and 30 minutes after injection. The injection should be deferred if pre-injection lung function is less than 70% of the predicted value for that patient.
• Patients should be monitored for at least 30 minutes after immunotherapy.

Practice tips
• Immunotherapy should be considered in patients with asthma who have documented allergen sensitisation. This decision should be made in consultation with allergy specialists or medical specialists trained in allergy. Important factors in this decision include the patterns of allergen sensitisation, the severity of disease, response to other therapy, the presence of other allergic disease and likelihood of adherence.
• Immunotherapy should be performed according to standard protocols under specialist guidance to reduce risks of adverse events.
• Immunotherapy should not be regarded as an alternative to established forms of prophylactic therapy. (For further information see Prevention of Asthma)
• Asthma needs to be monitored prior to, during, and after immunotherapy.

Sublingual immunotherapy in asthma (SLIT)

Sublingual immunotherapy (SLIT) is a promising alternative to SIT because it may be better tolerated and less invasive. It is not currently reimbursed through the Pharmaceutical Benefits Scheme (PBS).
• Although SLIT has shown therapeutic benefits in allergic rhinoconjunctivitis, consistent effects in asthma in terms of lung function, symptom scores and medication scores have not been reported.
• A number of issues need to be determined before SLIT can be considered as an acceptable therapeutic strategy in asthma, including: the relative efficacy of SLIT and SIT; the optimal dose and administration; the role of SLIT in allergy, asthma and asthma prevention.
• The clinical benefits of SLIT may be maintained for up to 10 years after discontinuation.
• SLIT has a good safety profile in adults and children. No serious adverse effects have been reported. Mild or moderate adverse effects (including local itching, asthma, urticaria, and rhinoconjunctivitis) were reported in less than 10% of children in long-term studies.


Practice tips
• SIT and SLIT should only be done in consultation with an allergy specialist or a medical specialist trained in allergy.
• SLIT is self-administered, increasing the risk of poor adherence and dosing errors.

Anti-IgE monoclonal antibody therapy

Monoclonal antibodies to the Fce domain of IgE have been assessed extensively in clinical trials. These forms of therapies show good short-term clinical benefits and are well tolerated, but do not show any sustained benefits after therapy is discontinued.
• A recent Cochrane review examined 8 randomised controlled trials of Anti-Fce (omalizumab) in asthma (2037 patients), and observed a significant reduction in IgE (98–99%), symptoms, bronchial hyper-reactivity, medication usage and asthma exacerbations. Omalizumab was well tolerated.
• Anti-IgE monoclonal antibody therapy is expensive and its role in asthma management in Australia is not well defined.

For more information, see Drugs and devices.

Other immunomodulatory strategies currently under investigation

New techniques aim to achieve increased and sustained clinical benefits, while reducing the risk of adverse effects compared with established immunomodulatory therapy options.
• Allergen-gene vaccination, allergen-peptide immunotherapy, cytokine therapy and refined microbial agents remain experimental.
• The place of food supplements with immunomodulatory characteristics (e.g. probiotics, antioxidants and omega-3 fatty acids) remains unclear. For more information, see Complementary and alternative medicine in asthma.
### Ongoing care

#### SUMMARY OF PRACTICE POINTS

<table>
<thead>
<tr>
<th>Assess asthma control regularly</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inappropriate dose increases of reliever or preventer should be avoided in young children with transient infant wheeze or intermittent viral-induced wheezing, because it is often not possible to eradicate this symptom completely using either SABA (I) or ICS (II) treatment.</td>
<td>I &amp; II</td>
</tr>
<tr>
<td>In children, attempts to completely eradicate cough by increasing asthma medication may result in over-treatment and increase the risk of adverse effects.</td>
<td>III-2</td>
</tr>
<tr>
<td>In the absence of other asthma symptoms, cough should not be used as a marker of asthma control.</td>
<td>III-2</td>
</tr>
<tr>
<td>Isolated persistent cough is rarely asthma.</td>
<td>III-2</td>
</tr>
<tr>
<td>In children, symptoms are as reliable as PEF for monitoring asthma control.</td>
<td>II</td>
</tr>
<tr>
<td>In Aboriginal and Torres Strait Islander people with asthma and a past history of pneumonia, careful assessment is needed to rule out additional respiratory illness.</td>
<td>IV</td>
</tr>
</tbody>
</table>

#### Identify and avoid trigger factors

- Take a careful history to establish possible allergic triggers within the person’s home or work environment. | ✓ |
- Consider skin prick tests or RAST to identify specific immunoglobulin (Ig)E antibodies to a suspected trigger. | ✓ |
- Reassure patients that food is not a common trigger of asthma. | II |
- Warn patients who use complementary medicines that echinacea and royal jelly can precipitate life-threatening anaphylaxis in predisposed individuals with asthma. | IV |
- A trial of acid suppression therapy may be worthwhile if GORD is suspected. However, asthma control does not predictably improve if reflux is treated (I). | ✓ & I |

#### Prevent exacerbations

- In the absence of bacterial infection, antibiotics are not indicated during viral respiratory tract infections in people with asthma. | I |
- Influenza vaccination is recommended in patients with severe persistent asthma (in addition to other indications). | ✓ |
- Influenza vaccination is very unlikely to cause asthma exacerbations. | I |
- Pneumococcal vaccination is indicated in patients with asthma who also have chronic bronchitis, emphysema, or require long-term systemic corticosteroid use (in addition to other indications). | ✓ |

#### Provide asthma self-management education

- All adults with asthma should be offered self-management education that involves a written action plan, self-monitoring and regular medical review. This approach can improve asthma control. | I |
- Asthma action plans enable patients to manage their asthma exacerbations appropriately (when provided with other self-management education). | I |
- Provide active training in inhaler technique and reinforce regularly, to maximise correct use of inhalation devices. | II |
- Provide confidential health care for adolescents, where appropriate. | II |
This chapter deals with the ongoing management of asthma as a chronic disease.

For information on initiating therapy and using drugs appropriately, see Principles of drug therapy.

For information on managing exacerbations and acute asthma see Managing exacerbations and Acute asthma.

Once the initial presenting asthma attack has been managed, or in any patient with a diagnosis of asthma, the ongoing aims of asthma management are:

• to minimise the symptoms
• to maximise lung function and maintain best lung function at all times
• to identify trigger factors
• to minimise unwanted effects of medication, in order to:
  • minimise morbidity and risk of life-threatening asthma episodes
  • prevent permanent impairment of lung function
  • maximise quality of life.

Achieving these aims requires individual assessment and careful ongoing reassessment of the treatment plan, and is most likely when there is a close working relationship between a committed doctor, an interested pharmacist and an informed patient. Other health professionals, such as Aboriginal health workers, asthma educators and practice nurses, also play important roles.

**Assess asthma control regularly**

Effective long-term management of asthma requires continual reassessment of control.

For information on assessment during an acute episode, see Acute asthma.

**Practice points**

• Inappropriate dose increases of reliever or preventer should be avoided in young children with transient infant wheeze or intermittent viral-induced wheezing, because it is often not possible to eradicate this symptom completely using either SABA (I) or ICS (II) treatment.
• In children, attempts to completely eradicate cough by increasing asthma medication may result in overtreatment and increase the risk of adverse effects. (III-2)
• In the absence of other asthma symptoms, cough should not be used as a marker of asthma control. (III-2)
• Isolated persistent cough is rarely asthma. (III-2)
• In children, symptoms are as reliable as PEF for monitoring asthma control. (II)
• In Aboriginal and Torres Strait Islander people with asthma and a past history of pneumonia, careful assessment is needed to rule out additional respiratory illness. (IV)
Asthma history checklist for new patients

When assessing a new patient with a prior asthma diagnosis, **confirm the diagnosis**. The history should include:

- current symptoms
- pattern of symptoms
- asthma history
- trigger factors
- current management (including complementary therapies)
- recent changes in management and the effect of those changes
- adverse effects of drug treatments
- pattern of a typical exacerbation
- impact of asthma on occupation
- atopic disorders
- general health and drug treatments
- patient’s (or carer’s) knowledge and ability to self-manage.

Assessment of control

The day-to-day management of asthma, including adjustment of medications, closely depends on ongoing assessment of asthma control.

- Asking the same questions at each consultation (Table 1) is a useful way to compare asthma control between consultations.
- At any general practice consultation (even if asthma was not the primary reason for the visit), the GP can take the opportunity to ascertain whether asthma control is good, fair or poor (Table 2).
- Check for signs, symptoms or history that suggest a high-risk patient. See **Identify high-risk patients**.
- At each pharmacy visit for asthma medication, the pharmacist can ask about asthma control and reinforce the importance of regular reassessment.

**Note:** the term “GP” denotes the patient’s primary health care provider. In some cases this role may be performed by other health professionals including Aboriginal health workers or nurses.

Using validated asthma control tools

Asthma control tools are a useful aid in measuring a patient’s asthma status and are designed to support patient consultations. Selection of an asthma control tool will depend on a number of criteria and the context in which you work. The International Primary Care Respiratory Group (IPCRG) has produced a useful, succinct User’s Guide to currently available asthma control tools, this is available at: [www.theipcrg.org](http://www.theipcrg.org). Two of the asthma control tools recommended in the IPCRG guide are:

- The Asthma Score (known as the Asthma Control Test in the USA). This is a validated tool for people 12 years and over and has 5 items (available at [www.asthmascore.com.au](http://www.asthmascore.com.au)).
- The Asthma Control Questionnaire (Juniper ACQ). This is a validated tool for people 16 years and over and has 6 items (available at [www.juniper@quoltech.co.uk](http://www.juniper@quoltech.co.uk)).

### Table 1. Questions to ask at every consultation

1. On average, how often are you \( ^1 \) woken by your asthma during the night?
2. On average, how bad are your asthma symptoms when you wake up in the morning?
3. In general, how limited are you in your activities because of your asthma?
4. In general, how much shortness of breath do you experience because of asthma?
5. In general, how often did you wheeze over the past few weeks or since the last visit?
6. On average, how many puffs of short-acting beta2 agonist (SABA) reliever (e.g. **Ventolin**) do you use each day?
7. How much work/school has been missed due to asthma?

\( ^1 \) Or patient, where carers asked on a patient’s behalf

### Table 2. Assessment of asthma control

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Good</th>
<th>Fair</th>
<th>Poor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime symptoms</td>
<td>None</td>
<td>&lt; 3 doses/week</td>
<td>≥ 3 doses/week</td>
</tr>
<tr>
<td>Night-time symptoms</td>
<td>Not woken</td>
<td>≤ 1 night/week</td>
<td>&gt; 1 night/week</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Normal</td>
<td>Normal</td>
<td>Restricted</td>
</tr>
<tr>
<td>Exacerbations</td>
<td>None</td>
<td>Mild, infrequent</td>
<td>Moderate, severe frequent</td>
</tr>
<tr>
<td>Missed school/work due to asthma</td>
<td>None</td>
<td>None</td>
<td>Any</td>
</tr>
<tr>
<td>Reliever use ( ^1 )</td>
<td>None</td>
<td>&lt; 3 doses/week</td>
<td>&lt; 3 doses/week</td>
</tr>
<tr>
<td>( ^{\text{**FEV1}} ), ( ^{\text{**FEV1/FVC}} )</td>
<td>Normal</td>
<td>≥ 90% personal best</td>
<td>&lt; 90% personal best</td>
</tr>
<tr>
<td>( ^{\text{**PEF}} )</td>
<td>Normal</td>
<td>≥ 90% personal best</td>
<td>&lt; 90% personal best</td>
</tr>
</tbody>
</table>

\( ^1 \) Does not include one dose per day for prevention of exercise-induced symptoms. **Applicable to adults and older children. Lung function parameters are not appropriate measures of asthma control in younger children.

---

适应症为: 国家哮喘协会澳大利亚 2006. 非营利性复制用于患者咨询或教育目的只允许。
Assessment of severity

During an acute asthma episode, assessment of the severity of presenting signs and symptoms is crucial to effective management. See Acute asthma.

This chapter is concerned with ongoing assessment of chronic asthma. Asthma severity applies to the underlying disease (not the severity of an acute attack) and should be assessed when asthma is stable.

- Overall asthma severity does not predict the severity of acute episodes (e.g. many children with intermittent asthma may have severe acute attacks).
- Asthma is classified as intermittent, mild persistent, moderate persistent or severe persistent.
- In a patient with newly diagnosed asthma, classification is based on the frequency and duration of respiratory symptoms, and the degree of persistent airflow limitation prior to the commencement of preventive treatment. Once asthma is well controlled, one of the best ways to judge severity is to determine the type and dose of medication needed to maintain good control (Table 3), taking into account the occurrence of severe acute episodes and effect of asthma on daily activities.4

For information on the assessment of asthma severity in a patient with untreated asthma, see Diagnosis and classification of asthma in adults and Diagnosis and classification of asthma in children.

Severity versus control

It can be helpful to make a distinction between a patient’s degree of asthma control at any given time, and the severity of the underlying disease.5

- Control is assessed as good, fair or poor according to recent reliever requirement, recent symptom frequency, nocturnal asthma, recent exacerbations, unplanned visits, current lung function (in adults and older children), and sometimes airway hyperresponsiveness.5
- Severity is classified according to the minimum amount of medication and intensity of interventions required to achieve and sustain good asthma control.5

In practice, severity and control interact (Table 3). For example, asthma symptoms might be well controlled by appropriate treatment, despite underlying severe asthma. (Of the two concepts, control is most relevant to the day-to-day care of a patient with asthma in general practice or in the pharmacy.)

Table 3. How asthma pattern, severity and control interact

<table>
<thead>
<tr>
<th>Asthma pattern</th>
<th>Asthma control</th>
<th>Medication required*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent</td>
<td>Good</td>
<td>Adults</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reliever PRN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Children with infrequent intermittent asthma Reliever PRN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Children with frequent intermittent asthma Reliever PRN and Low-dose ICS or oral leukotriene receptor antagonist or inhaled cromone</td>
</tr>
<tr>
<td>Mild persistent</td>
<td>Good</td>
<td>Adults</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reliever PRN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Children</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Reliever PRN and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Low-dose ICS or</td>
</tr>
<tr>
<td></td>
<td></td>
<td>leukotriene receptor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>antagonist or inhaled</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cromone</td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>Good</td>
<td>Low–moderate ICS ±</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LABA</td>
</tr>
<tr>
<td>Severe persistent</td>
<td>Good-fair</td>
<td>Moderate–high ICS +</td>
</tr>
<tr>
<td></td>
<td>(poor if very severe)</td>
<td>LABA ± other</td>
</tr>
</tbody>
</table>

Table 4. ICS dose equivalents: what is meant by low, medium and high daily doses?

<table>
<thead>
<tr>
<th>Daily ICS dose</th>
<th>CIC*</th>
<th>BDP–HFA**</th>
<th>FP**</th>
<th>BUD**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>80–160 mcg</td>
<td>100–200 mcg</td>
<td>100–200 mcg</td>
<td>200–400 mcg</td>
</tr>
<tr>
<td>Medium</td>
<td>160–320 mcg</td>
<td>200–400 mcg</td>
<td>200–400 mcg</td>
<td>400–800 mcg</td>
</tr>
<tr>
<td>High</td>
<td>320 mcg and above</td>
<td>Over 400 mcg</td>
<td>Over 400 mcg</td>
<td>Over 800 mcg</td>
</tr>
</tbody>
</table>

ICS: inhaled corticosteroid; LABA: long-acting beta2 agonist; CIC: ciclesonide; BDP–HFA: beclometasone dipropionate; FP: fluticasone propionate; BUD: budesonide

* ex actuator dose
** ex valve dose

Adapted from Juniper AF et al, 19942; Cockcroft DW, Swystun VA, 19966

*Sample medication regimens typically required to achieve the corresponding degree of control (not all medications listed). For information on the use of each class of medication, see Principles of drug therapy.

**If a non-corticosteroid therapy is desired, LTRA maybe an alternative treatment.
Assessment of asthma in adults and adolescents

Current asthma control should be re-checked regularly, to ensure that the medication regimen is optimal. See Questions to ask at every consultation and Asthma history checklist for new patients.

Objective lung function tests

Accurate measurement of respiratory function is necessary to assess and manage asthma.

Spirometry

- Changes in clinical improvement correlate poorly with lung function measured by spirometry. Therefore symptoms alone are not sufficient to assess lung function.
- Spirometry is the preferred method for assessing airflow in the office during ongoing asthma review, as well as in diagnosis.
- Successive measurements before and after bronchodilator are used to determine whether adequate asthma control has been attained. The presence of a significant bronchodilator response (at least 12% increase in FEV1) might suggest sub-optimal control.
- During down-titration of medication, pre-bronchodilator spirometry is sufficient for ensuring that an individual’s best lung function is maintained.

For more information on spirometry see Diagnosis and classification of asthma in adults and Diagnosis and classification of asthma in children.

Peak expiratory flow

Regular measurement of peak expiratory flow (PEF) using a portable peak flow meter is useful for some people to monitor their asthma, as an aid to self-management.
- Measurement of PEF is useful to assess effects of occupational triggers. See Occupational asthma.
- Single PEF measurements are not adequate for use in routine asthma management by doctors.

Assessment of asthma in children

In children who have commenced regular preventive treatment, regularly assess response to treatment and ongoing asthma control.

- In preschool-aged children, assess response to initial treatment carefully because transient infant wheeze and intermittent viral-induced wheezing may not respond to treatment with either a SABA reliever or inhaled corticosteroid (ICS) preventer. In this group, the appropriate next step may be cessation of preventive treatment, rather than dose escalation.
- In children with typical asthma symptoms, the long-term aim of treatment is always to achieve and maintain good control with the minimum effective dose of preventive treatment.

Symptoms

Cough

- Cough is a major symptom in some children with asthma, and can be a useful marker of asthma control provided that symptoms of airflow limitation (wheeze, breathlessness, exercise limitation) are also taken into account. During asthma exacerbations and in stable asthma, severity of cough indices does not correlate well with the severity of other asthma indices. Isolated persistent cough is rarely due to asthma. The presence of cough does not reliably predict the onset of an asthma exacerbation in all children.
- Nocturnal wheezing and dyspnoea on waking are more reliable than cough in assessing asthma pattern and severity.
- Because the pathophysiology of cough is different from that of other asthma symptoms, cough may not respond as readily to preventive asthma treatment. Accordingly, attempts to eradicate cough completely by increasing asthma medication may result in over-treatment and potential adverse effects. Conversely, disappearance of cough does not mean that other objective lung function measures have improved.
- Indoor heating alone significantly improves cough in children with asthma, even where the asthma does not improve.

For more information on the interpretation of recurrent non-specific cough in the diagnosis of asthma in children, see Diagnosis and classification of asthma in children.
Wheeze and breathlessness

- Breathlessness on exertion is not specific to asthma and is a poor marker for the diagnosis of asthma in children.23
- Chest tightness and wheezing have a higher negative predictive value and positive predictive value than cough, dyspnoea or exercise testing as markers of airflow limitation.24
- Recurrent wheeze in children less than 2 years old may not respond to SABA treatment, so care is needed when using this symptom as an outcome marker in young children.6
- Viral-induced wheezing episodes are often not completely abolished with preventive asthma treatment.7 Occasional breakthrough episodes may be acceptable in a child with no symptoms between episodes.

Practice tips

In children, avoid potentially hazardous dose escalation by:

- gauging symptom control by taking into account the overall symptom profile (wheeze, breathlessness and exercise limitation).
- avoiding making an assessment of asthma control based on any single symptom, particularly cough, in isolation.
- checking that spirometry or peak flow measurement is performed properly before using the result to guide treatment.
- carefully assessing initial response to treatment to determine whether a lack of response indicates poorly controlled asthma or an alternative diagnosis (particularly in pre-school children).
- understanding that the total absence of isolated cough, or of wheeze associated with viral respiratory tract infections, may not be an achievable and is not necessarily an appropriate treatment goal.

Physical examination

At each review:

- assess objective signs of chronic airflow limitation (wheeze on auscultation when ‘well’, chest deformity, hyperinflation). Caution: Be aware that (1) these signs of chronic respiratory disease are not specific to asthma; and (2) their absence does not reliably rule out asthma.
- check growth. Poor growth may be a sign of either under-treated asthma or an unwanted effect of ICS.

Objective lung function tests

Spirometry

Spirometry is the preferred method for ongoing assessment of response to treatment in children old enough to perform the spirometry manoeuvre properly (≥ 7 years). Correct technique is essential to obtain the most reliable information from spirometry.


Portable peak flow meters

- Remember that PEF is effort-dependent and that a submaximal effort invalidates the reading. This is a significant problem in children, and those under 7 or 8 years old may not be able to perform the test reliably.
- Beware of over-treatment based on a poorly performed PEF reading. Check the patient’s technique in the surgery and/or pharmacy, using the same meter each time.
- Regular peak flow monitoring has a limited role in children and should be reserved for those with moderate to severe persistent asthma, particularly if they have difficulty perceiving worsening airflow limitation.
- Several recent randomised controlled trials assessing guided self management in childhood asthma have failed to show any advantage of PEF-based monitoring over symptom monitoring.25–27
**Ongoing review**

Pre-planned, structured review of asthma – as distinct from opportunistic or on-demand review – is associated with reduced rates of exacerbations, improved symptom control and fewer days absent from work or school.28–34

In adults, the timing of asthma review depends on disease control and pattern (e.g. annually in those with good control, at change of season in those with seasonal triggers, or every few weeks where closer monitoring and medication adjustments are needed).

In children, asthma should be reviewed:
- every 3 months in children who require regular preventive medication
- every 12 months in children with intermittent asthma, providing that they have a comprehensive written asthma action plan.

**Identify patients with high-risk asthma**

The following characteristics identify adults, adolescents or children who are potentially at risk from life-threatening asthma, and indicate the need for close follow-up:
- Frequent visits to emergency department or GP with acute asthma, or hospital admission for asthma in previous 12 months.
- Requirement for three or more medications to control symptoms or need for continuous or multiple courses of oral corticosteroids.
- A history of admission to an intensive care facility or a previous near-fatal attack.
- Night-time attacks, especially associated with severe chest tightness or ‘choking’.
- Failure to perceive asthma symptoms when spirometric values are decreased.
- Excessive reliance on inhaled bronchodilators.
- Denial of asthma as a problem, or other overt psychosocial problems.
- Inadequate treatment or poor adherence to treatment, especially in teenagers or young adults.
- Asthma triggered by aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs) including, much less frequently, cyclo-oxygenase (COX)-2 inhibitors (e.g. celecoxib).
- Poor asthma control:
  - persistent morning dips in PEF (< 60% recent best)
  - marked (> 25%) diurnal variation in PEF in adults.
- Among young children, boys may have higher risk for life-threatening asthma than girls.

Consider also anaphylaxis risk, e.g. in patients with immediate hypersensitivity to foods, especially nuts.

**Care of the high-risk patient**

- Review at least once every 3 months.
- Assess lung function at each visit using spirometry with bronchodilator response.
- Refer to a consultant respiratory physician, if possible.
- The primary care provider must communicate well with the patient’s consultant, pharmacist and carer.
- Review asthma management plan and written action plan, with contact telephone numbers.
- Ensure that the patient or carer always has a supply of beta2 agonists and oral corticosteroids for emergency management.
- Identify and address psychosocial issues.
- Discuss and resolve any barriers to adherence to treatment plan.
- Involve an asthma educator, if available.35,36

**Identify and avoid trigger factors**

- Take a careful history to establish possible allergic triggers within the person’s home or work environment. ✓
- Consider skin prick tests or RAST to identify specific immunoglobulin (Ig)E antibodies to a suspected trigger. ✓
- Reassure patients that food is not a common trigger of asthma. (II)
- Warn patients who use complementary medicines that echinacea and royal jelly can precipitate life-threatening anaphylaxis in predisposed individuals with asthma. (IV)
- A trial of acid suppression therapy may be worthwhile if GORD is suspected. ✓ However, asthma control does not predictably improve if reflux is treated. (I)
- Exercise-induced asthma may be an indication of under-treatment. ✓
Trigger factors may be allergic or non-allergic, and include:

- allergens
- respiratory infections (See Prevent exacerbations)
- exercise (See Exercise-induced asthma)
- drugs
- foods
- gastro-oesophageal reflux
- smoking (See Smoking and asthma)
- air pollutants
- occupational factors (See Occupational asthma)
- temperature change.

Explain to patients how to avoid triggers where possible (with the exception of exercise). Continued exposure to allergens and other trigger factors may lead to worsening of asthma. Avoidance of trigger factors may improve asthma.

**Allergens**

Take a careful history to establish possible allergic triggers within the person’s home or work environment.

Skin prick tests or radioallergosorbent tests (RAST) can identify specific immunoglobulin (IgE) antibodies to a suspected trigger. Commonly recognised triggers include house dust mite, pollens, animal danders and, possibly, moulds.

For information on asthma and anaphylaxis risk, see Asthma and Allergy.

**Dust mite**

The house dust mite is the most common allergen source in humid towns and cities. Dust mite is difficult to eradicate and the effectiveness of attempts to reduce exposure to this allergen is controversial.

**When to consider an allergy consultation**

Consultation with a physician specialising in allergy may be helpful for patients with:

- asthma in conjunction with anaphylactic features
- sudden unexplained episodes of asthma
- known or suspected hypersensitivity to foods
- signs and symptoms that raise suspicions about allergic factors, but in whom an allergic cause is not obvious
- asthma in conjunction with other problems, especially hay fever and skin disorders
- persistent unstable asthma with hospital presentations.

For more information on allergic asthma triggers and allergy tests, see Asthma and Allergy.


**Drugs**

When prescribing or advising a patient on any new medication for another medical problem, ask the person to report any deterioration in asthma control.

Examples of medications that may cause or worsen asthma include:

- beta-adrenergic blocking agents, either oral or in eye drops
- aspirin and other NSAIDs
- cholinergic agents (e.g. carbachol, pilocarpine)
- cholinesterase inhibitors (e.g. pyridostigmine)
- some complementary medicines (e.g. echinacea, royal jelly) can precipitate life-threatening anaphylaxis in predisposed individuals with asthma.

For more information on drugs that may worsen asthma, see Drugs and devices.
Foods

Foods are not common triggers of asthma. The isolated occurrence of asthma symptoms without cutaneous or gastrointestinal symptoms is rare.\(^{38}\)

In some individuals, foods can trigger acute episodes of wheeze, due to:\(^{39,40}\)
- food allergies (IgE-mediated hypersensitivity) – most commonly to nuts, shellfish, milk and eggs, or
- non-allergic food hypersensitivity (mechanisms not involving the immune system).

No single food, food chemical or additive (e.g. metabisulfite) acts as a trigger in all people with asthma, and only a few people with asthma have hypersensitivity to additives.\(^{41}\) Sensitivity to some additives previously suspected as triggers (e.g. monosodium glutamate) has proved to be very uncommon when tested under controlled conditions.\(^{42,44}\)

For more information, see Diet and asthma.

Gastro-oesophageal reflux

Up to 40% of adults with asthma experience gastro-oesophageal reflux disease (GORD). A trial of acid suppression therapy may be worthwhile if GORD is suspected. However, asthma control does not predictably improve if reflux is treated.\(^{43,44}\)

For more information on GORD, see Other comorbidities.

Air pollutants

Indoor and outdoor pollutants can trigger asthma exacerbations in some people with asthma. Patients can be advised to avoid exposure to:
- smog
- bushfire smoke/fallout
- formaldehyde, nitrogen dioxide and volatile organic compounds (e.g. household cleaners, paint fumes)
- environmental tobacco smoke.


Air environment

A drop in air temperature at night can trigger asthma and may be prevented by heating the bedroom at night.

The use of humidifiers is not recommended for people with asthma.

Prevent exacerbations

Practice points

- In the absence of bacterial infection, antibiotics are not indicated during viral respiratory tract infections in people with asthma. (I)
- Influenza vaccination is recommended in patients with severe persistent asthma (in addition to all other indications). ✓
- Influenza vaccination is very unlikely to cause asthma exacerbations. (I)
- Pneumococcal vaccination is indicated in patients with asthma who also have chronic bronchitis, emphysema, or require long-term systemic corticosteroid use (in addition to all other indications). ✓

Exacerbations occur due to various causes, which may which may contribute to variation in inflammatory features,\(^{45}\) clinical patterns,\(^{46}\) and response to treatment.\(^{47}\) Different causal factors may interact to exacerbate asthma, e.g. allergen exposure and sensitisation with viral infection,\(^{48}\) or thunderstorms and allergen exposure and sensitisation.\(^{49}\)

Infections

- In children, infection with common respiratory viruses is the most common trigger for acute exacerbations of asthma.\(^{50}\) The common cold virus is implicated in approximately 80% of exacerbations in children,\(^{50}\) whereas influenza infection probably accounts for only a small proportion of asthma exacerbations.
- On current evidence, the use of antibiotics is not recommended in the absence of bacterial respiratory infection.\(^{51}\)
- Contemporary guidelines do not recommend the use of antibiotics during acute asthma exacerbations in children or adults, either routinely or when there is low suspicion of bacterial infection.\(^{2,52,53}\)

Influenza vaccine

Australian guidelines recommend annual influenza vaccination for people with severe persistent asthma (including those who require frequent hospitalisation), for Aboriginal and Torres Strait Islander people over 50 years old, and for everyone over 65 years.\(^{54}\)
- Despite previous concerns that influenza vaccine might precipitate asthma attacks, current evidence suggests that there is a very low risk of an asthma
exacerbation immediately following influenza vaccination.55-57
• Although recommended in guidelines, influenza vaccination has not been shown to protect against asthma exacerbations.57,58

Pneumococcal vaccination
Australian guidelines recommend pneumococcal vaccination for all children, Aboriginal and Torres Strait Islander people over 50 years old, and for everyone over 65 years old.54,59
• Asthma is a risk factor for pneumococcal infection and vaccination should be considered based on individual risk.60
• Current Australian guidelines recommend pneumococcal vaccination in those with pulmonary disease but do not specify asthma as a condition necessitating routine pneumococcal vaccination.54,59
At present there is not strong evidence that everyone with asthma should received pneumococcal vaccination.61
• Pneumococcal vaccination is indicated in patients with asthma who also have the additional risk factors of chronic bronchitis, emphysema, or require long-term systemic corticosteroid use.62

Monitoring deterioration in asthma control
See Managing exacerbations and Asthma action plans.

Provide asthma self-management education

Practice points
• All adults with asthma should be offered self-management education that involves a written* action plan, self-monitoring and regular medical review. This approach can improve asthma control. (I)
• Asthma action plans enable patients to manage their asthma exacerbations appropriately (when provided with other self-management education). (I)
• Provide active training in inhaler technique and reinforce regularly, to maximise correct use of inhalation devices. (II)
• Provide confidential health care for adolescents, where appropriate. (II)

*Those who do not read English require an action plan written in their first language or careful verbal training.

Education helps patients gain confidence, skills and motivation to control their asthma.63 This process should begin at the time of the diagnosis and continue in all subsequent consultations. The aim is to enable the person to manage their own (or their child’s) asthma effectively, in order to:
• achieve the best possible health (physical and psychological)
• reduce the need for unplanned GP visits, emergency room visits and hospital admissions
• enjoy the greater freedom and convenience of being confident to manage most changes in asthma control.

In adults, asthma health status is improved and unplanned visits for asthma are markedly reduced by asthma self-management education programs that involve all of the following key components:63
• monitoring of asthma control, using either PEF or symptoms
• a written asthma action plan
• regular medical review.

Understanding the uses of asthma medications and training in the correct use of inhaler devices are important components of self-management.

When working with a patient to enable them to manage their asthma effectively, health professionals should:
• acknowledge that objective measures of lung function alone do not adequately reflect the person’s asthma status, and that subjective symptoms and quality of life are important considerations in treatment
• encourage the patient or carer to become more involved in decision making, yet continue to take professional responsibility for patients’ medical care
• consider patients’ individual circumstances
• provide information in a suitable language and medium (e.g. written, audiotape in first language, illustrated flip books) or refer patients to Asthma Foundations with these resources. See Information resources for patients.

Not all patients are prepared, or suited, to take significant responsibility for self-management, but may nevertheless benefit from general information and education about their asthma. These people may, in time, begin to take more responsibility for management of their condition.
‘Self-management’ goes beyond giving asthma information

Self-management for people with chronic disease conditions involves the individual working in partnership with carers and health professionals so that they can:

• understand their condition and be aware of various treatment options
• negotiate a plan of care and review the plan
• engage in activities that protect and promote health
• monitor and manage the symptoms and signs of the condition
• manage the impact of the condition on physical functioning, emotions and interpersonal relationships.

Structured self-management programs

Effective structured self-management programs have been developed for adults with asthma. These are based on successful generic chronic disease self-management programs, with the addition of asthma-specific components (e.g. training in optimal use of medications, detection and management of worsening asthma control, identifying and dealing with asthma triggers).

• Training programs that give patients the ability to adjust their asthma medications using a written asthma action plan appear to be more effective than other asthma self-management program.

• In adults with asthma, providing information as the only form of education does not result in any clinically important improvements in health care utilisation or lung function.

• Approximately one emergency department visit for asthma is prevented for every 22 patients who undertake an intensive self-management program that reinforces adherence and aims to reduce exacerbations.

For availability of structured self-management programs, contact the Asthma Foundation in your state or territory. See Information resources for patients.

The essential elements of asthma self-management education are:

• written information about asthma (See Self-management education checklist in Appendices)
• self-monitoring of symptoms and/or PEF
• a written asthma action plan. See Asthma action plans.
• regular review by a doctor, asthma educator or nurse.

This involves reassessment of medications and current asthma control, reinforcement of trigger avoidance behaviours and lifestyle changes if warranted, feedback to the patient about how well they are controlling their asthma and how control can be improved.

Training in correct use of inhaler devices

✓ Practice tip

Check whether patients are using inhaler devices correctly by having them demonstrate their inhaler technique during the consultation.

For all patients using inhaled asthma medications, health care professionals should provide verbal instructions and a physical demonstration of correct technique. Active training in inhaler technique is necessary for correct technique.

• Training in correct inhaler use by a doctor, asthma educator (where available), nurse or pharmacist can markedly improve technique in children and adults.

• Correct technique should be regularly reinforced when prescribing, dispensing or advising on inhalers.

Asthma action plans

Asthma action plans help the patient or carer to recognise and respond appropriately to worsening asthma. They are individually tailored according to the pattern of the adult or child's asthma, written and provided for the patient to keep as a wallet-sized card or paper sheet. The plan must be carefully explained to the patient. Patients who do not read need the information explained in a way they can remember.

An individual’s plan includes:

1. Guidance for identifying signs of worsening control, which can include the following:
   • increasing frequency or severity of symptoms, especially waking at night with asthma
   • the need for increasingly frequent doses of bronchodilator
   • failure of bronchodilator to completely relieve symptoms
   • falling PEF
   • increasing PEF variability.

2. Clear instructions for how to respond to any given change in asthma control, which may include:
   • altering medications or doses
   • consulting the GP
   • attending an emergency care facility.

Written asthma action plans are equally effective when based on PEF monitoring or symptom diaries.
• When PEF is used, the action plan should be based on personal best rather than on predicted values.73
• Inclusion of PEF monitoring in the asthma action plan can be beneficial for people with more severe or difficult-to-control asthma,27,74 and those with poor perception of airflow limitation. See Asthma in the elderly.

When developing the asthma action plan, the patient’s individual preferences for decision-making processes should be consulted. Many patients with moderate-to-severe asthma prefer their doctor to make decisions about medication changes.75

Templates for asthma action plans are available from the National Asthma Council Australia and Asthma Foundations, and are included in general practice management software. See the asthma action plan in Appendices.

Asthma action plans for adults
For information on drug therapy, see Principles of drug treatment in adults.

In adults, the use of individualised asthma action plans (also called self-management plans):63,66
• reduces absences from work
• reduces hospital admissions
• reduces emergency presentations to general practice
• reduces SABA use
• improves lung function.

Asthma action plans for children and adolescents
For information on drug therapy in children, see Principles of drug treatment in children and adolescents.

• When choosing the delivery device, take into account the patient’s age, the drugs prescribed and (for older children and adolescents) the child’s own preferences.
• Encourage parents to give the school a copy of the student’s asthma action plan (essential for school trips).
• Some schools request the GP to complete an asthma first aid plan.
• Guidelines for the care of children with asthma while at school (the Asthma-Friendly Schools program) have been developed for Australian schools. For more information contact the Asthma Foundation in your territory; free call 1800 645 130.

Self-management for adolescents
For young people to learn to manage their asthma effectively, it is essential that they and their parents become actively involved in working with the GP and other health professionals.
• Health professionals can help foster adolescents’ engagement in their asthma care by encouraging their parents to recognise that the young person needs to develop independence. A useful approach is to see the young person alone for part of the consultation and maintain confidentiality where appropriate,76 as well as listening to parents and supporting their roles.77
• Parents can promote and respect the patient’s autonomy while continuing to supervise treatment and monitor asthma control.76 Young people with chronic disease are more likely to adhere to medication within a well-functioning and supportive family, even up to their early twenties.79,80 Adolescents with chronic disease regard overly strict, controlling and critical parental behaviour as a hindrance to self-management,81 e.g. a parent’s constant reminders to take medication are counter-productive when interpreted by a young person as unwarranted nagging.
• Peer support groups76,82 and school-based self-management programs are effective for young people with chronic disease.

Opportunistic asthma education
• Primary health care service visits and pharmacy visits provide opportunities for informal asthma education and review of patients’ or carers’ ability to manage asthma.
• Emergency hospital admissions have also been used to engage patients for asthma education programs. Among patients who have been hospitalised for asthma exacerbations, as few as two individualised education sessions can be effective over the short term in improving patients’ knowledge of asthma, adherence to medications and inhaler technique, and in reducing further emergency admissions.83

Self-management should be part of a comprehensive care program
• Effective management of asthma requires not only pharmacological management, but also patient knowledge of asthma and an effective working relationship between the patient and the GP.
• Self-management involves the person participating as an active partner in asthma care, but it is not an alternative to medical care. Severe or life-threatening attacks are more likely to occur in patients with inadequate medical supervision.63
• Regular recall and reassessment by a health professional is essential for objective assessment of lung function by spirometry, review of symptoms, review of patient-initiated changes to therapy, checking of inhaler technique, review of the asthma action plan, review of trigger factors and how to avoid them, and ongoing asthma education to reinforce the treatment plan.

Encourage people with asthma to:
• understand the basic pathophysiology and natural history of asthma
• understand the different roles of their reliever and preventer medications, and symptom controllers, if prescribed
• take continuing responsibility for their asthma
• make appropriate changes to medication when necessary
• contact their doctor or other appropriate health care professional if they have concerns or queries regarding their asthma management
• attend when free of acute exacerbations for regular review. (Frequency of review will depend on the pattern of asthma and the response to treatment.)
• bring their inhaler device to the consultation so that their inhaler technique can be checked
• have their asthma action plan updated every 12 months.

Information resources for patients
There is a wide range of resources available to help you educate people with asthma and their carers about asthma self-management.
• The National Asthma Council Australia website contains all the NAC publications in printable form and is linked to a range of reputable national and international asthma and respiratory websites: www.nationalasthma.org.au.
• The Asthma Foundations are not-for-profit community-based organisations providing a variety of services to people with asthma, their carers and health professionals. These include telephone advice lines staffed by trained advisers, support groups, asthma camps and swimming classes for children, training programs and asthma educators for individual or group education. You may wish to refer asthma patients to your local Foundation for assistance in education and for support. To contact the Asthma Foundation in your area, call 1800 645 130.
• Asthma education materials designed for Aboriginal and Torres Strait Islander people are available from the Asthma Foundation of the Northern Territory (www.asthmant.org.au).

Troubleshooting

Practice points
• When asthma is inadequately controlled despite apparently appropriate treatment, increase medication doses only after careful reassessment of the diagnosis, triggers, inhaler technique and adherence. ☑
• If possible, avoid the use of multiple types of inhalation devices to deliver asthma medications, because this increases risk of poor inhaler technique. (III-2)

When a person’s asthma control is suboptimal despite apparently appropriate treatment, assess the possible causes of the problem carefully before increasing medication doses or changing the regimen.

Troubleshooting checklist
• Review medical history – consider comorbid. conditions and alternative diagnoses.
• Reassess triggers (including medications, allergens, irritants).
• Check inhaler technique by observation.
• Assess adherence to the treatment plan.
• Review treatment plan.

Reassess triggers
Consider whether exposure to known triggers or new triggers may have increased since asthma symptoms were well controlled. See Identify and avoid triggers.

Medications
Check whether the person has begun taking any new medications that may affect asthma, including prescription drugs, non-prescription drugs or complementary medicines.
Allergens and irritants

- Check if there may have been a recent increase in exposure to environmental triggers.
- Check if lifestyle changes that may be associated with exposure to triggers (e.g. change in office air conditioning, new home, spending more time in smoky bars/clubs, spending weekends with a new partner who is a smoker or has pets).

Review the history

- Consider whether the person may have another condition that is affecting asthma control or mimicking asthma, including other causes of breathlessness.
- In preschool-aged children a range of wheezing phenotypes may occur, and other conditions may mimic asthma symptoms.
- Consider gastro-oesophageal reflux. See Other comorbidities.
- Consider other lung lesions. Chest X-ray or repeated spirometry may be required.
- Consider cardiac disease. Other investigations including echocardiography may be warranted if heart failure cannot be ruled out. Referral to a cardiologist may be indicated.
- Carefully review cough in children
  - In children, a persistent or recurrent cough in the absence of wheeze may be wrongly attributed to asthma.\(^{84}\)
  - The diagnosis of asthma in children should not be made on the basis of cough alone. ‘Cough-variant’ asthma is rare and its existence is now questioned.\(^{20,85}\)
  - In children with asthma in whom cough is a prominent symptom, review the response to treatment carefully, so as to avoid long-term ineffective treatment with ICS or ICS dose escalation.\(^{17}\)

  ✓ Practice tips

  All health professionals can advise people with asthma and their carers:
  - not to begin taking any new medications (prescription, non-prescription, complementary), until they have checked with their doctor or pharmacist whether the new medicine may affect their asthma
  - to visit the same GP or health service consistently, where possible
  - to buy medicines from one pharmacy, where possible.

Review self-management proficiency

Asthma control is improved when patients receive appropriate education in asthma management.\(^{63}\)

- Assess and reinforce the person’s understanding of the roles and appropriate use of inhaled medications
- Assess and reinforce the person’s understanding of their asthma action plan. Update the asthma action plan if necessary. See Provide self-management education.

Assess attitudes

- Does the person understand and accept the diagnosis of asthma?
- Is the person unwilling or embarrassed to use their inhaler in public?

Reassess inhaler technique

- Before considering an increase in doses or additional drugs, inhaler technique should be reassessed by direct observation, especially if the asthma is poorly controlled. Correct inhaler technique should be reinforced regularly. For more information on training in correct inhaler technique, see Provide self-management education.
- Avoid using more than one type of inhalation device to deliver asthma medications within a patient’s regimen. Incorrect inhaler technique is more common when more than one type of inhaler device is used.\(^{86}\)

✓ Pharmacy practice tip

Pharmacists can check and reinforce correct inhaler technique when dispensing devices.

Check adherence

- Most people with asthma use their medication as prescribed when they are symptomatic, as there is an immediate connection between taking medication and the relief of symptoms. For the same reason, adherence to reliever medication tends to be greater than adherence to preventive medication. Once symptoms resolve, continued adherence becomes

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
increasingly difficult for many people with asthma. Whenever asthma control is poor in the face of apparently adequate treatment, consider the issue of poor adherence. Assessment of adherence requires an open, non-judgemental approach.

- Emphasise the benefits of regular preventer use.

**When to refer to a specialist**

**Adult patients**

Consultation with a specialist respiratory physician is recommended in the following situations:

- a life-threatening acute asthma attack
- poor self-management ability requiring intensive education*
- poor perception of worsening symptoms
- poor adherence to treatment plan*
- uncertain diagnosis
- no response to therapy
- abnormal lung function persisting when the symptoms are apparently controlled
- need for frequent courses of oral corticosteroid
- daily ICS dose requirement greater than BDP–HFA/FP 800 mcg or BUD 1600 mcg or CIC 320 mcg
- unacceptable side-effects from medication
- chest X-ray abnormalities
- possible occupational causes and aggravators
- when detailed allergy assessment is indicated
- allergic bronchopulmonary aspergillosis suggested by cough, plugs of mucus, resistant symptoms, positive pathology testing for *Aspergillus*.

*Refer to an asthma educator.

**Children and adolescents**

Referral for paediatrician or respiratory paediatrician review should be considered for children who have any of the following:

- Uncertain diagnosis
- Poor response to treatment
- Significant school absence
- Frequent hospitalisations or a life-threatening episode
- Persisting lung function abnormalities
- Moderately high ICS doses required to maintain control:
  - more than 250 mcg FP/HFA–BDP or more than 400 mcg BUD in children under 5 years old
  - more than 500 mcg FP/HFA–BDP or more than 800 mcg BUD in children over 5 years old
- Frequent courses of oral corticosteroids
- Systemic corticosteroid side effects or other unacceptable side effects from medication
- Other complicating medical or psychosocial issues.

**Organising your practice for effective asthma management**

**Practice points**

- Appropriately trained nurses can effectively undertake a substantial proportion of tasks related to asthma management, including the asthma review. (II)
- Proactive care in general practice, combined with an active recall system, increases the use of written asthma action plans, reduces airway hyperresponsiveness and decreases the rate of emergency visits for asthma in children. (II)
- Assessment for exposure to environmental tobacco smoke should be undertaken and specific interventions considered where exposure is identified. (IV)

**Practice tips**

- Ensure your medical records system allows you to readily identify all patients with asthma.
- Set up a recall/reminder system for regularly reviewing each person’s asthma.
- Tag (electronically or physically) files for all patients with asthma, to prompt opportunistic assessment when patients present with other problems or for routine script renewal.
- Computerised record systems can be very effective for tracking patients for follow-up, and for identifying patients on inappropriate treatment regimens (e.g. high-dose ICS without LABA).
- Make use of resources for patients and health professionals provided by the National Asthma Council Australia, Asthma Foundations, your Division of General Practice.
- Consider setting up an asthma clinic program in which asthma educators work with GPs.
The organisation of primary health care systems for managing chronic diseases influences the quality of clinical care. Asthma outcomes are improved by:

- organising a follow-up consultation soon after emergency hospital visits for acute asthma
- setting up systems that enable proactive, routine clinical review, as distinct from opportunistic or unscheduled review
- offering patients effective self-management education. See Provide self-management education.

These strategies rely on efficient information management systems.

- Primary health care services need to set up and regularly maintain a register of all patients with a diagnosis of asthma, so that regular review and education can be offered.
- The use of computers to support clinical care (e.g. for decision support, accessing discharge summaries, case finding and clinical guidelines) is associated with better quality of clinical care in patients with chronic diseases including moderate-to-severe asthma.
- Participation of administrative staff in maintaining registers and recall systems improves the practice’s ability to deliver effective asthma care.

Asthma clinics

The practice of conducting regular general practice asthma clinics (dedicated sessions for patients with asthma, involving routine review by the GP and/or practice nurse) is becoming popular in the UK. Limited clinical trial data suggest that participation in asthma clinics can reduce nocturnal asthma symptoms and promotes the use of peak flow meters.

Considerations for organisation of asthma review

- In children with moderate-to-severe asthma, the use of written asthma action plans can be increased, airway hyperresponsiveness improved and emergency visits for asthma reduced through pre-planned general practice visits for asthma review, combined with an active recall system.
- Not all patients are willing to attend pre-arranged visits. Patients’ perception of the severity of their asthma and attitudes to follow up influence whether they attend planned asthma reviews scheduled outside periods of exacerbation.
- Asthma reviews carried out by telephone may be as effective as face-to-face consultations.
- Follow-up consultation within 30 days after emergency hospital attendance with acute asthma is associated with reduced risk of further acute episodes within the first 90 days of the initial emergency visit. Ensuring this review depends on effective hospital discharge protocols, including prompt communication to the GP, and systems within the primary health care facility to enable efficient tracking and recall of patients.
- In adults with asthma, continuity of care is the most important factor contributing to a good patient-doctor relationship. Patients’ satisfaction with their doctor is a key factor contributing to adherence to maintenance preventer medication.

Recent changes to the Asthma 3+ Visit Plan for patients with moderate to severe persistent asthma include an annual cycle of care rather than three consultations within four months. Patients on the new Asthma Cycle of Care would need a minimum of two asthma-related consultations within a year, with the second visit planned at the first.

The role of the practice nurse

Potential roles for practice nurses in the care of patients with asthma include:

- conducting asthma review
- performing spirometry
- providing information and self-management education, especially in the areas of correct use of medications, inhaler technique and training to use their action plans as developed by the GP
- case finding
- managing recall and reminder systems
- coordination/administration for services to which special Medicare Chronic Disease Management item numbers apply.

Specialised training is necessary for practice nurses to be effective in asthma care.

Asthma educators can effectively undertake a substantial proportion of tasks related to asthma management, including the asthma review. Overall, the content of asthma management interventions, the level of training undertaken by health care professionals providing the care, and the quality of interaction between patient and health professional appear to be more important than who provides the care.

- In health care services where a practice nurse conducts the asthma review, asthma outcomes are similar to those seen when a doctor conducts the review.
• Ongoing asthma management by appropriately trained nurses produces similar asthma outcomes to standard care by GPs.
• Clinical trials and audits evaluating asthma care provided by nurses within primary care have reported mixed outcomes ranging from no significant improvements compared with standard GP care to increased patient contact with the practice and improved control of asthma symptoms.
• Asthma clinics led by nurses and supported by doctors have been found to be a convenient structure for providing comprehensive asthma care, but their benefits are not well established.
• Asthma care by hospital nurses can effectively support general practice. Where collaboration between hospital and GP services is established, provision of asthma education for patients and clinical support for GPs by skilled specialist respiratory nurses can reduce unscheduled care for asthma in patients who have previously required emergency hospital visits.

Practice tips
Some GPs find these routines convenient for incorporating spirometry into the consultation:
• The GP performs the pre-bronchodilator test and administers SABA first, then completes the history and examination during the 10 minutes' wait, then does the post-bronchodilator test last.
• The practice nurse performs spirometry before the consultation.
• Education on inhaler and spacer technique can be given during the 10-minute wait.
• Patients are requested to advise the receptionist whenever a consultation is for asthma review, and the receptionist routinely schedules a longer appointment.

The role of the community pharmacist
Successful asthma management and the best outcome for the patient are most likely to be achieved when there is a close working relationship between the doctor, pharmacist and patient.
• Asthma care programs involving community pharmacists have been shown to improve quality of life and improve some clinical outcomes. Report benefits include reduced symptoms, improved perception of asthma control, increased PEF, reduced absences from work or school, reduced SABA use, reduced emergency room visits and medical visits, improved asthma knowledge, reduced medication costs and improved quality of life, compared with usual care.
• Pharmacists can effectively train patients in correct inhaler technique.
• With appropriate training in asthma care, pharmacists can improve asthma outcomes by educating patients about medications, triggers and self-monitoring, providing an asthma action plan, monitoring outcomes and adherence, and consulting with the patient's doctor to achieve asthma prescribing guidelines.

Ongoing monitoring and advice in the pharmacy
• Pharmacists are sometimes in a position to advise people with symptoms suggestive of undiagnosed asthma to visit their GP for full assessment.
• Pharmacists see patients regularly to dispense medications and can take the opportunity to check that asthma is well controlled and that the person is using appropriate doses of medications, and to reinforce correct use of medications and inhaler devices. If there is any suggestion of suboptimal control or need for further asthma education, the pharmacist can refer the person to their GP.
• Whenever prescribed, non-prescription or complementary medicines are requested by people with asthma, the pharmacist has the opportunity to check safety.
• Pharmacists can help patients quit smoking by providing information and supplying smoking cessation products. By mentioning smoking-associated risks to all parents of children with asthma, pharmacists can reinforce health messages about maintaining a smoke-free environment.

Home Medicines Review
• A Home Medicines Review can be conducted by a specially accredited pharmacist after referral by the patient's GP, with the aim of optimising drug management.
• The visit involves a comprehensive interview about the person's medications, and covers issues such as adherence, actual usage of medicines, concurrent use of complementary medicines, knowledge of asthma, giving information, assessing adverse effects and potential drug interactions.
• The pharmacist sends a written report to the GP for discussion with the patient.
Team Care Arrangements

- A pharmacist can qualify as a member of a chronic disease care team in Team Care Arrangements under the Chronic Disease Management Medicare items introduced 1 July 2005, where the pharmacist provides ongoing treatment or services to the patient (other than just providing routine dispensing or dispensing-related services). 104
- A GP might identify a need for a Home Medicines Review while coordinating Team Care Arrangements for a patient. A pharmacist conducting a Home Medicines Review might also recognise that a patient would benefit from a GP Management Plan or Team Care Arrangement.

Demographic considerations: organising your practice to suit your patients

The elderly

- Incorporate respiratory assessment (and asthma review for those with known asthma) in Aged Care Health Assessments and Comprehensive Medical Assessments in nursing homes.
- GPs who visit residential aged care facilities should be aware of asthma-related issues affecting the elderly, including under-diagnosis and difficulties with medications and monitoring asthma control.
- The use of Home Medicines Review is especially useful for older patients with multiple comorbidities and complex medication regimens.
- Practice nurses can undertake home spirometry for house-bound elderly patients.

For more information, see Asthma in the elderly.

Aboriginal and Torres Strait Islander patients

- When developing an asthma care service appropriate for Aboriginal and Torres Strait Islander people, health care providers should refer to the framework developed by the National Aboriginal and Torres Strait Islander Health Council for the Australian Health Ministers’ Advisory Council105 (available at www.health.gov.au).
- When designing health services for Aboriginal and Torres Strait Islander people, include a system for effective monitoring of accessibility of the service and effectiveness of asthma care.106
- The involvement of Aboriginal and Torres Strait Islander health workers within local community-controlled health services, and other primary healthcare settings, has been the basis of effective models of chronic disease care.105,107

Special considerations for Health Assessments

- People with asthma and a past history of pneumonia should be carefully assessed for additional respiratory illness, especially bronchiectasis, COPD and obstructive sleep disorders.108,109
- Assessment for exposure to environmental tobacco smoke should be undertaken and specific interventions considered where exposure is identified. Tobacco smoking rates are high in Aboriginal and Torres Strait Islander people, compared with non-indigenous populations.110
- Pneumococcal vaccination and annual influenza vaccine is recommended for all Aboriginal and Torres Strait Islander people aged 50 years and over.

Rural and remote regions

For information about asthma programmes in your area, contact The Australian College of Rural and Remote Medicine, your Division of General Practice or the Asthma Foundation in your state or territory.

Culturally and linguistically diverse groups

Some ethnic subgroups experience higher hospital admission and exacerbation rates.111 Contact the Asthma Foundation in your state or territory for help in overcoming linguistic difficulties and cultural isolation. Contact the Telephone Interpreter Service on 131 450 for a free interpreter service. Telephone and onsite interpreters can be pre-booked. See www.immi.gov.au/tis.

Economic status

Lower socio-economic status has been linked with poorer asthma outcomes, compared with higher socio-economic status.112

When setting up practice systems for asthma management, consider cost issues for patients, e.g. routinely offering treatment options that minimise out-of-pocket expenses, or making available information about payment and support options.
Smoking and asthma

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief smoking cessation advice from doctors delivered opportunistically during routine consultations improves quit rates.</td>
<td>I</td>
</tr>
<tr>
<td>Set up a system to identify and document tobacco use in all patients. This can almost double the rate of clinician intervention and improves cessation rates among patients.</td>
<td>II</td>
</tr>
<tr>
<td>Smoking cessation advice from all health professionals is effective in increasing quit rates.</td>
<td>I</td>
</tr>
<tr>
<td>Follow-up is effective in increasing quit rates.</td>
<td>I</td>
</tr>
<tr>
<td>Relapse prevention advice can reduce relapse rates.</td>
<td>II</td>
</tr>
<tr>
<td>Telephone call-back counselling services (e.g. Quit program) are effective in assisting smokers who are ready to quit.</td>
<td>II</td>
</tr>
<tr>
<td>Nicotine replacement therapy and bupropion sustained release are effective in helping motivated people to quit smoking.</td>
<td>I</td>
</tr>
<tr>
<td>Regardless of the treatment setting, all forms of nicotine replacement therapy (e.g. gum, transdermal patches, nasal spray, inhaler, sublingual tablets) approximately double quit rates at 5- to 12-month follow-up, compared with placebo.</td>
<td>I</td>
</tr>
<tr>
<td>In smokers who are highly nicotine-dependent, combinations of different forms of nicotine replacement therapy are more effective than one form alone.</td>
<td>II</td>
</tr>
<tr>
<td>Bupropion sustained release is effective in smoking cessation.</td>
<td>I</td>
</tr>
<tr>
<td>Acupuncture or hypnotherapy are ineffective in assisting smoking cessation.</td>
<td>I</td>
</tr>
<tr>
<td>Offer pneumococcal vaccination to all smokers.</td>
<td>✔</td>
</tr>
</tbody>
</table>

Smoking ranks second after being overweight as a major cause of premature death and illness among Australians.¹ About one in two regular smokers dies of a smoking-related disease.²

People with asthma have even more reason than those without asthma to avoid smoking. In addition to the known adverse health effects of smoking in the general community, the fact that cigarette smoking worsens asthma and reduces the effectiveness of medication warrants serious efforts by health professionals and patients to eradicate smoking.

**Smoking rates in Australia**

About 19% of males and 16% of females over 14 smoke daily.³

- Smoking rates are highest among people in their twenties: approximately one in four people aged 20–29 years smokes.³
- Approximately 11% of teenagers (14–19 years) smoke daily or weekly.³

Although fewer Australians are smoking than in the past, smoking rates are now disproportionately high in particular groups.

- Smoking rates are directly proportional to level of socioeconomic disadvantage. Of all demographic groups, smoking rates are highest among young men (18–34 years) living in the most socioeconomically disadvantaged areas.⁴
- Indigenous people aged 18 years or over are twice as likely as non-Indigenous people to be current smokers.⁵ Approximately half (49%) of Indigenous Australians aged 15 years and over smoke daily.⁵ In comparison, approximately 17% of non-Indigenous Australians aged 14 and over smoke daily.³
- Approximately 50%–80% of people with mental illness smoke.⁶ ⁷
• Approximately 17% of women report smoking while pregnant. Rates of smoking during pregnancy are particularly high among teenagers (42%) and Aboriginal and Torres Strait Islander women (52%).

Exposure to environmental tobacco smoke among children

• Over 600,000 Australian children are exposed to tobacco smoke inside the home. Smoking inside the home is reported in approximately 12% of households with children under 14 years.
• Among children with asthma aged 14 years or less, 41% live in a household with one or more regular smokers and therefore risk potential exposure to cigarette smoke. In comparison, approximately 38% of children of the same age group without asthma live with one or more smokers.
• Among children with asthma, those from areas of relative socioeconomic disadvantage are most likely to be living with a regular smoker.

Active smoking rates among people with asthma

Despite advice against smoking, people with asthma are no less likely to smoke than those without asthma.
• Overall, 26% of people with current asthma smoke: 27% of males (compared with 28% of males without asthma) and 25% of females (compared with 21% of females without asthma).
• Among people with asthma, those aged 18–34 years are most likely to smoke.

Effects of smoking on asthma

Effects of active smoking in people with asthma

People with asthma who smoke suffer additional morbidity.
• People with asthma who smoke experience more respiratory symptoms, worse asthma control, more airway inflammation, an inferior short-term response to inhaled corticosteroid treatment, and an accelerated decline in lung function than those who do not smoke.
• Smoking increases the risk of chronic obstructive pulmonary disease (COPD). In people with asthma, this can result in overlapping of COPD symptoms with those of asthma, which can delay the diagnosis of COPD and complicate management. For more information see COPD and asthma.

Emerging evidence suggests that smoking is causally associated with the development of adult-onset asthma.

Effects of exposure to environmental tobacco smoke in people with asthma

Exposure to environmental tobacco smoke has been shown to contribute to a variety of health problems in children: respiratory infections, middle ear infections, onset and worsening of wheezing and asthma, impaired lung function, eye and nose irritation, low birth weight, sudden infant death syndrome and increased use of medical services.
• In utero exposure to cigarette smoke is associated with reduced lung function and increased risk of respiratory illnesses including wheeze and asthma.
• The link between exposure to environmental tobacco smoke in early childhood and increased risk of respiratory illnesses, including asthma, has been well documented in epidemiological studies.
• Exposure to environmental tobacco smoke aggravates pre-existing asthma in children, and contributes to approximately 8% of childhood asthma in Australia (46,500 children). Children of mothers who smoke heavily (more than 10 cigarettes per day) are most at risk.
• In adults, exposure to cigarette smoke has been shown to increase asthma morbidity.

Make the car and home a smoke-free zone

Health professionals and parents should aim for a smoke-free home for all children, including those with asthma. Where children with asthma live in households with at least one smoker, an absolute ban on smoking inside the home has been shown to reduce levels of nicotine in the air and to reduce levels of cotinine (a breakdown product of nicotine) in children’s urine.

Mechanisms for effects of smoking on asthma

Increased bronchial hyperresponsiveness and increased levels of atopy are the mechanisms most likely to explain the onset or worsening of asthma due to tobacco smoke exposure.
• Among children with asthma, those with mothers who smoke show greater variability in peak expiratory flow rates over a 24-hour period and have airways that are more sensitive to adverse pharmacological or physical stimuli.
Smoking and asthma

• Rates of atopy are higher among children of smoking mothers than children of non-smoking mothers.\(^{21,22}\)
  Biological markers of atopy (e.g. elevated serum immunoglobulin levels) are observed more frequently in children exposed to environmental tobacco smoke than in unexposed children.\(^{23}\)
• In utero exposure to environmental tobacco smoke may adversely modify neonatal immune responses.\(^{24,55}\)
• Importantly, cigarette smoking also reduces the efficacy of inhaled corticosteroid (ICS) treatment in people with asthma. Following ICS treatment, smokers appear to achieve less benefit than non-smokers according to asthma exacerbations, lung function measures and inflammatory markers.\(^{26,27}\)

Clinical interventions to help patients quit smoking

Advice given during primary care consultations, even if brief, can effectively influence patients to quit (Figure 1). Information and advice should be tailored to the patient’s circumstances and preferences and may involve directing the person to Quitline, formal referral to a Quit program, or counselling by a GP, practice nurse or pharmacist.


Lifescripts materials (assessment tool, guide to helping patients quit smoking, prescription) are available through Divisions of General Practice or at: www.adgp.com.au or www.health.gov.au.

Advice and counselling

Health professionals can effectively help people to quit smoking.
• Brief advice from a doctor to stop smoking, given during a routine consultation, achieves a small increase in the proportion of patients who successfully quit smoking during the following 6–12 months.\(^{28}\)

Practice points

• Brief smoking cessation advice from doctors delivered opportunistically during routine consultations improves quit rates. (I)
• Set up a system to identify and document tobacco use in all patients. This can almost double the rate of clinician intervention and improves cessation rates among patients. (II)
• Smoking cessation advice from all health professionals is effective in increasing quit rates. (I)
• Follow-up is effective in increasing quit rates. (I)
• Relapse prevention advice can reduce relapse rates. (II)
• Telephone call-back counselling services (e.g. Quit program) are effective in assisting smokers who are ready to quit. (II)
• Offer pneumococcal vaccination to all smokers

Pneumococcal vaccination

All smokers should be offered pneumococcal vaccination, regardless of their readiness to quit, because of the increased risk of invasive pneumococcal disease.\(^{29}\)
Drug therapy

Nicotine replacement therapy and bupropion sustained release are effective in helping motivated people to quit smoking. Smokers using pharmacotherapy should be encouraged to use it for a sufficient period (8 weeks with nicotine replacement therapy and at least 7 weeks with bupropion sustained release). Regardless of the treatment setting, all forms of nicotine replacement therapy (e.g., gum, transdermal patches, nasal spray, inhaler, sublingual tablets) approximately double quit rates at 5- to 12-month follow-up, compared with placebo. In smokers who are highly nicotine-dependent, combinations of different forms of nicotine replacement therapy are more effective than one form alone. Combination nicotine replacement therapy should be offered if patients are unable to remain abstinent or continue to experience withdrawal symptoms using one type of therapy. Bupropion sustained release is effective in smoking cessation, both alone and in combination with nicotine replacement therapy. Combination treatment with bupropion and nicotine patch should be considered for those who have not achieved smoking cessation during an adequate trial of either therapy alone. Blood pressure should be monitored during treatment.

Other strategies for quitting

- Introducing smoking restrictions into the home can assist quitting smoking successfully.
- Acupuncture or hypnotherapy are ineffective in assisting smoking cessation.

Information for smokers is available at www.quitnow.info.au.

Figure 1. GP time expenditure for smoking cessation

Supportive organisational infrastructure

- 0 minutes Quit rate* doubled

Brief intervention

- <1 minute Quit rate trebled

Moderate intervention

- 2-5 minutes Quit rate increased 4-fold

Intensive intervention

- > 5 minutes Quit rate increased 5-7 fold

Adapted from Lifescripts Division Kit

*Estimated increase in quit rates over 12 months among patients attending the practice, compared with not applying the intervention.
Public policy

Australian public policy initiatives are vitally important to reduce smoking rates and, consequently, exposure to environmental tobacco smoke. Smoking policy should be informed by documented evidence for influences on smoking uptake and quitting.

Influences on smoking uptake

- Parental smoking strongly influences the likelihood of children taking up regular smoking, while keeping the home smoke-free reduces children’s chances of taking up the habit.
- Addiction to nicotine at age 15 years carries a high risk of continuing to smoke beyond 35 years. Smoking at age 35 years carries a cumulative 50% of premature death and reduced healthy years of life.
Promotion of smoking cessation

Public policy initiatives that are effective in reducing smoking include the following:

- Comprehensive bans on tobacco advertising and promotion, prominent warning labels, restrictions on smoking in public places, and increased access to nicotine replacement treatments\textsuperscript{36,38}
- Tax increases that raise the price of cigarettes\textsuperscript{36,38}
- Improvements in the quality and extent of information on smoking-related health risks.

The Royal Australasian College of Physicians and the Royal Australian and New Zealand College of Psychiatrists advocate the adoption and implementation of a range of policies on smoking by Australian state and territory governments.\textsuperscript{36} These include:

- encouraging medical practitioners and healthcare institutions to diagnose smoking as a major health condition
- supporting smoking cessation programs by subsidising professional services and pharmaceuticals
- supporting medical schools in systematically training medical students in smoking cessation interventions.

For a comprehensive list of RACP policies, see The Royal Australasian College of Physicians and the Royal Australian and New Zealand College of Psychiatrists. Tobacco policy: using evidence for better outcomes. Sydney: RACP and RANZCP, 2005.\textsuperscript{36}
Chronic obstructive pulmonary disease (COPD) and asthma

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Always attempt to make a firm diagnosis in patients with airflow limitation, because the natural history and optimal management of COPD and asthma differ significantly.</td>
<td>✔</td>
</tr>
<tr>
<td>Consider COPD in all at-risk patients because early diagnosis and treatment improves outcomes.</td>
<td>II</td>
</tr>
<tr>
<td>Consider COPD in patients who have other smoking-related diseases.</td>
<td>I</td>
</tr>
<tr>
<td>Consider COPD in all smokers and ex-smokers older than 35 years.</td>
<td>II</td>
</tr>
<tr>
<td>Set up a system to alert you to consider COPD in at-risk patients: smokers, elderly patients, patients with a diagnosis of asthma who do not respond to treatment as expected.</td>
<td>✔</td>
</tr>
<tr>
<td>Investigate breathlessness in patients at risk for COPD, even when the patient attributes it to ageing or poor fitness.</td>
<td>✔</td>
</tr>
<tr>
<td>Investigate further whenever a patient mentions consistent sputum production or persistent cough.</td>
<td>✔</td>
</tr>
<tr>
<td>The diagnosis of COPD is based on the demonstration of airflow limitation that is not fully reversible.</td>
<td>✔</td>
</tr>
<tr>
<td>Anticholinergic bronchodilators and SABAs are effective in managing symptoms.</td>
<td>I</td>
</tr>
<tr>
<td>Tiotropium and LABAs provide sustained relief of symptoms, improve exercise performance and reduce the frequency of severe exacerbations in moderate-to-severe COPD.</td>
<td>I</td>
</tr>
<tr>
<td>Short-course oral corticosteroids reduce the severity and duration of exacerbations.</td>
<td>I</td>
</tr>
<tr>
<td>Long-term use of oral corticosteroids is not recommended.</td>
<td>I</td>
</tr>
<tr>
<td>Pulmonary rehabilitation reduces dyspnoea, anxiety and depression, improves exercise capacity and quality of life and may reduce hospitalisation rates in patients with COPD.</td>
<td>I</td>
</tr>
<tr>
<td>Pulmonary rehabilitation reduces dyspnoea, anxiety and depression, improves exercise capacity and quality of life and may reduce hospitalisation rates in patients with COPD.</td>
<td>I</td>
</tr>
<tr>
<td>Inhaled corticosteroids should be reserved for those with severe COPD and frequent exacerbations.</td>
<td>✔</td>
</tr>
<tr>
<td>Assessment of walking distance and the patient’s report of symptomatic improvement are appropriate guides to effectiveness of therapy.</td>
<td>✔</td>
</tr>
</tbody>
</table>

Chronic obstructive pulmonary disease (COPD) is a progressive, disabling disease characterised by symptoms of breathlessness during physical activity and/or daily cough with or without sputum, airway inflammation and airflow limitation that is not fully reversible.

- The precursor conditions that most commonly lead to COPD are small-airway narrowing (with or without chronic bronchitis) and emphysema caused by smoking.¹

- Chronic bronchitis, emphysema and asthma overlap within COPD (Figure 1).
- COPD is characterised by intermittent acute exacerbations of symptoms (sputum production, breathing difficulties or both), which are usually due to respiratory tract infection, and can be significantly disabling.
Consider the possible diagnosis of COPD in all those who are at risk (e.g. smokers and ex-smokers, elderly patients) or who show airflow limitation that is not fully reversible.

An understanding of COPD is relevant to asthma management because:

• asthma and COPD have different prognoses and require different management\(^1^{,2}\)
• both asthma and COPD can overlap or coexist
• asthma and COPD have many common features, and may be difficult to distinguish\(^3\)
• both asthma and COPD are under-diagnosed in the elderly\(^4^{,5}\)


In practice, it is useful to distinguish between the diagnoses of asthma and COPD in each individual patient’s case, because there are important differences between the optimal management for each of these conditions.

**Prevalence**

Chronic obstructive pulmonary disease occurs almost exclusively in adults, and prevalence increases with age.\(^10\)

• Approximately 50% of all smokers develop some airflow limitation, and 15%–20% will develop clinically significant disability.\(^1\)
• Approximately 9%–12% of people over 45 years old have symptomatic COPD\(^11^{,12}\)
• The number of people with unacknowledged symptoms of chronic, poorly reversible airflow limitation has been estimated to be at least two to three times higher than the number of those with diagnosed COPD.\(^13^{,14}\)

**Risk factors**

Tobacco smoking is the most important risk factor for COPD.

Other risk factors include:

• exposure to environmental tobacco smoke
• exposure to dusts and chemicals in the workplace
• exposure to indoor biomass fuel smoke in people from some traditional cultures
• a strong family history of COPD
• recurrent respiratory infections in childhood
• atopy
• alpha-1-antitrypsin deficiency (uncommon).
The possibility of COPD should be considered in:
• all patients with any other smoking-related disease
• all smokers and ex-smokers over 35 years old.

Diagnosis

Practice points

- Always attempt to make a firm diagnosis in patients with airflow limitation, because the natural history and optimal management of COPD and asthma differ significantly. ✓
- Consider COPD in all at-risk patients because early diagnosis and treatment improves outcomes. (II)
- Consider COPD in patients who have other smoking-related diseases. (I)
- Consider COPD in all smokers and ex-smokers older than 35 years. (II)
- Set up a system to alert you to consider COPD in at-risk patients: smokers, elderly patients, patients with a diagnosis of asthma who do not respond to treatment as expected. ✓
- Investigate breathlessness in patients at risk for COPD, even when the patient attributes it to ageing or poor fitness. ✓
- Investigate further whenever a patient mentions consistent sputum production or persistent cough. ✓
- The diagnosis of COPD is based on the demonstration of airflow limitation that is not fully reversible. ✓

✓ Practice tip
Consider the possibility that a patient may have both COPD and asthma.

The differential diagnosis of COPD includes:
• other respiratory diseases (e.g. bronchiectasis, interstitial lung diseases)
• non-respiratory diseases (e.g. chronic heart failure, anaemia).

The diagnosis of COPD is based on the history, together with the demonstration of airflow limitation that is not fully reversible by performing spirometry before and after administration of a short-acting beta₂ agonist (SABA) bronchodilator.15,16

Clinical features of asthma that distinguish it from COPD are:17
• significant variation in airflow limitation between visits
• significant variation in airflow limitation with different treatments
• reversal of airflow limitation on spirometry in response to a short-acting bronchodilator.

However, asthma and COPD can be difficult to distinguish – especially in older adults, in whom respiratory symptom patterns are frequently non-specific. Misdiagnosis is common.15,18,19

Table 1. Symptoms of COPD

<table>
<thead>
<tr>
<th>Major symptoms</th>
<th>Other symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Breathlessness</td>
<td>• Chest tightness</td>
</tr>
<tr>
<td>• Cough</td>
<td>• Wheezing</td>
</tr>
<tr>
<td>• Sputum production</td>
<td>• Airway hyperresponsiveness</td>
</tr>
</tbody>
</table>

In advanced disease
• Fatigue
• Poor appetite
• Weight loss

History
The history should include:
• symptoms (Table 1)
• exposure to risk factors, including occupational history
• factors that may suggest new-onset asthma in an older person, including post-retirement exposure to new allergens and sensitisers, use of medications that may precipitate bronchoconstriction (e.g. nonsteroidal anti-inflammatory drugs, beta-adrenergic receptor antagonists). See Asthma in the elderly.

Physical examination
Physical examination is not a sensitive test for detecting mild-to-moderate COPD.1,20
• Wheezing is not an indicator of severity and is often absent in stable, severe COPD.
• The presence and severity of airflow limitation are impossible to determine by clinical signs.
• Chest overinflation (enlarged chest diameter, percussion hyper-resonance with loss of cardiac dullness, and reduced overall respiratory excursions) increases with worsening airflow limitation and emphysema.
Spirometry

Spirometry is the recommended method for confirming the diagnosis, assessing severity and monitoring COPD.

- The ratio of forced expiratory volume in one second (FEV₁) to vital capacity (VC) is a sensitive indicator of mild COPD.
- Peak expiratory flow (PEF) is not a sensitive measure of airway function in COPD.

Spirometry is indicated in patients with any of the following:

- unexplained breathlessness
- cough that is chronic (daily for two months) or intermittent and unusual
- frequent or unusual sputum production
- recurrent acute infective bronchitis
- risk factors (e.g. exposure to tobacco smoke, occupational dusts and chemicals, and a strong family history of COPD).

A degree of fixed airflow limitation is present if both the following are recorded 15–30 minutes after administration of SABA bronchodilator medication:

- The ratio of FEV₁ to forced vital capacity (FVC) is < 70% (< 0.70) and
- FEV₁ < 80% predicted.


Other tests

Biomarkers from induced sputum or breath condensates are currently being evaluated for diagnostic use, but are not yet suitable for clinical use.21–23

Special diagnostic considerations in the elderly

- Elderly patients with COPD may not become aware of cough and dyspnoea or report any symptoms until they have already become significantly disabled and lung function is moderately impaired (e.g. FEV₁ may be reduced to as little as 50% of predicted value).15
- Asthma and COPD can be clinically indistinguishable in elderly patients.3

For a suggested approach to distinguishing asthma from COPD in older patients, see Asthma in the elderly.

Practice tip

Supplemental oxygen given during an acute asthma episode may cause respiratory depression and arrest in a patient with comorbid COPD. For more information on the management of acute asthma, including respiratory arrest, see Acute asthma.

Management of COPD

Practice points

- Anticholinergic bronchodilators and SABAs are effective in managing symptoms. (I)
- Tiotropium and LABAs provide sustained relief of symptoms, improve exercise performance and reduce the frequency of severe exacerbations in moderate-to-severe COPD. (I)
- Short-course oral corticosteroids reduce the severity and duration of exacerbations. (I)
- Long-term use of oral corticosteroids is not recommended. (I)
- Pulmonary rehabilitation reduces dyspnoea, anxiety and depression, improves exercise capacity and quality of life and may reduce hospitalisation rates in patients with COPD. (I)
- Inhaled corticosteroids should be reserved for those with severe COPD and frequent exacerbations. ✓
- Assessment of walking distance and the patient’s report of symptomatic improvement are appropriate guides to effectiveness of therapy. ✓

The management of COPD must be tailored to the individual’s symptoms and needs, aiming to maximise quality of life.1,3 COPD is often complicated by comorbidities related to age and smoking history, such as ischaemic heart disease, chronic heart failure, diabetes mellitus, osteoporosis, sleep-disordered breathing, reduced ability to perform activities of daily living, physical deconditioning, depression, malnutrition, gastro-oesophageal reflux disease, lung cancer, and degenerative arthritis.

Current Australian and New Zealand ‘COPDX’ guidelines are based on the following goals:1

- C: confirm the diagnosis and assess severity
- O: optimise lung function
- P: prevent deterioration
- D: develop a support network and self-management plan
- X: manage exacerbations.
Smoking cessation is an important initial goal of COPD management, in order to reduce the rate of decline in lung function.24

Pulmonary rehabilitation is an important part of management. It reduces dyspnoea and fatigue, improves patients’ sense of control over their disease, and improves exercise capacity.25 No changes in spirometry are usually seen, despite the significant improvements in function, quality of life and symptoms.

Practice point
Assessment of walking distance and the patient’s report of symptomatic improvement are appropriate guides to effectiveness of therapy.

Drug treatment
Optimal pharmacological management of COPD differs significantly from that of asthma (Table 2).

Drug treatment for patients with COPD is based on the following principles:1

- **Inhaled bronchodilators** (short-acting beta₂ agonists and anticholinergic agents) are recommended for symptom relief. These agents:
  - are effective in the management of acute COPD exacerbations
  - may increase exercise capacity in the short term
  - are equally effective in COPD
  - may be better tolerated used in combination (compared with higher doses of either a SABA or anticholinergic agent used alone).

Although significant improvements in FEV₁ may not be seen in response to inhaled bronchodilators, patients may nevertheless experience symptomatic relief, increased exercise tolerance and improved ability to perform activities of daily living. Spirometric measurements are generally poor predictors of clinical improvement in response to bronchodilators in COPD.26

- **Long-acting inhaled bronchodilators** (salmeterol, eformoterol, tiotropium) provide sustained relief of symptoms, reduce the risk of exacerbations, and improve exercise capacity and health status in moderate-to-severe COPD. Current evidence suggests that tiotropium is more effective than either of the beta₂ agonists.27

- **Oral corticosteroids** are used short-term to manage acute exacerbations. Short-course oral corticosteroids:
  - reduce the severity of acute COPD exacerbations and speed recovery26
  - are not recommended for long-term use.
  - Inhaled corticosteroids (ICS) are generally reserved for patients with severe COPD and frequent exacerbations or those who have severe COPD with frequent exacerbations.

Table 2. **Major differences between COPD management and asthma management in adults**

<table>
<thead>
<tr>
<th>Asthma</th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular ICS treatment is recommended for patients of all ages with persistent asthma.</td>
<td>ICS are generally reserved for patients with severe disease and frequent exacerbations, or those who have shown improvement with ICS therapy.</td>
</tr>
<tr>
<td>Anticholinergic agents (e.g. tiotropium, ipratropium) are not used.</td>
<td>Tiotropium 18 mcg once daily improves dyspnoea, exacerbation rates, exercise capacity and health status.</td>
</tr>
<tr>
<td>Antibiotics are rarely indicated to manage exacerbations.</td>
<td>The use of antibiotics is often appropriate in the management of exacerbations.</td>
</tr>
</tbody>
</table>

Home oxygen therapy
Patients with COPD who have suspected chronic hypoxaemia should be assessed for home oxygen therapy.1

- Long-term continuous oxygen therapy is appropriate for patients with consistent hypoxaemia.
- Intermittent oxygen therapy may be considered for patients with hypoxaemia on exertion, those living in isolated areas or prone to sudden life-threatening episodes while awaiting emergency care, and those travelling by plane.
- Nocturnal oxygen therapy is required for patients with hypoxaemia during sleep, and should be considered in patients with daytime somnolence, polycythaemia or right heart failure despite adequate daytime oxygenation.

Exercise-induced asthma

Exercise-induced asthma and exercise-induced bronchoconstriction

- Exercise-induced asthma and exercise-induced bronchoconstriction are the terms commonly used to describe the transient narrowing of the airways that follows vigorous exercise in a dry environment. The term exercise-induced asthma is preferred when describing the response in clinically recognised asthmatics. Exercise-induced bronchoconstriction is the preferred term when describing the same response in people who do not have any other signs or symptoms of asthma. Both names however are frequently used to describe the response to exercise whether there is a clinical diagnosis of asthma or not.
- Exercise-induced asthma/exercise-induced bronchoconstriction is defined as a reduction in forced expiratory volume in one second (FEV₁) of 10% or more from the value measured before exercise.
- Exercise-induced asthma occurs in around 50–65% of people with asthma who are being treated with inhaled corticosteroids; a fall in FEV₁ of 30% or more is regarded as severe. Severe exercise-induced asthma is accompanied by arterial hypoxemia and lung hyperinflation, and requires medical attention.
- In Australia, exercise-induced bronchoconstriction has become increasingly recognised in school children and in elite athletes who have normal lung function.

Pathogenesis

- As inspired air is heated and humidified during exercise, water is evaporated from the airway surface. This process can cause exercise-induced asthma or...
The cooler and dryer the inspired air, the more severe the symptoms.

- Breathing warm humid air can prevent airway dehydration so that exercise does not provoke an attack of asthma.
- In elite athletes performing endurance summer sports, the levels of allergen they inhale during training may increase the risk of exercise-induced bronchoconstriction. In elite swimmers, environmental irritants arising from exposure to chlorine and its products is thought to contribute to the development of ‘twitchy’ airways, a feature of asthma.
- The pathogenesis of exercise-induced bronchoconstriction in winter athletes may relate to injury of the airway epithelial surface from conditioning large volumes of cold dry air. This is thought to lead to increased responsiveness of bronchial smooth muscle and subsequently symptoms of asthma and exercise-induced bronchoconstriction.

Impact on quality of life, asthma and sporting performance

Exercise-induced asthma/exercise-induced bronchoconstriction should not be allowed to interfere significantly with quality of life because treatment is so successful in preventing the problem.

- Because exercise-induced asthma/exercise-induced bronchoconstriction occurs after exercise, it should not provide a physiological limitation to exercise performance. However, there are some minor changes in lung function during exercise and, in competitive sport, these may contribute to performance.
- When exercise is performed within one hour of recovering from exercise-induced asthma/exercise-induced bronchoconstriction, approximately 50% of people become refractory and will have significantly less exercise-induced asthma/exercise-induced bronchoconstriction a second time.
- There is no evidence that exercise-induced asthma/exercise-induced bronchoconstriction impacts on asthma control but it may be regarded as a sign that asthma is not well controlled.

Detection

The best question to elicit a history of exercise-induced asthma/exercise-induced bronchoconstriction is: “Do you feel more breathless/wheezy/symptomatic five to ten minutes after you stop exercise than during exercise?”

- People without asthma will also get short of breath if they exercise hard enough, but the symptoms subside rapidly after the exercise stops.
- In someone with exercise-induced asthma/exercise-induced bronchoconstriction the symptoms get worse for the next 5 to 10 minutes before spontaneous recovery occurs over the next 30 minutes.
- Recovery from exercise-induced asthma/exercise-induced bronchoconstriction can be aided by the use of a bronchodilator to reverse the airway narrowing.
- Exercise-induced asthma/exercise-induced bronchoconstriction cannot be excluded on the basis of a negative test to inhalation of methacholine and histamine, particularly in people with normal spirometry. Leukotrienes and prostaglandins are considerably more potent in causing bronchial smooth muscle contraction than histamine and methacholine and they are the most important mediators of the airway narrowing provoked by exercise.

Practice points

- Respiratory symptoms during exercise are poor indicators of the presence of exercise-induced asthma, therefore, objective testing is recommended. (IV)
- Being physically fit can increase the intensity of exercise required to provoke exercise-induced asthma, although exercise-induced asthma can still occur. (I)

Assessment of lung function

Forced expiratory volume in one second (FEV1) is the best measurement to identify exercise-induced asthma/exercise-induced bronchoconstriction and to assess its severity. A peak flow meter can be used but the measurement has greater variability than FEV1. Repeated measurements are usually made before exercise and then repeated at least 3 times within 10 minutes of ceasing exercise.

When testing for exercise-induced asthma in the field, exercise should be:

- strenuous (> 85% maximum heart rate)
- preferably running
- performed for six minutes in children and eight minutes in adults
- take place in an environment that is dry or at least has less than 10 mg of water per litre of air (represented by a common room air temperature of 23°C with 40% humidity).
Several laboratory tests are used as surrogates for exercise to detect exercise-induced asthma/exercise-induced bronchoconstriction:

- Eucapnic voluntary hyperpnea: the major advantage of this test is that the duration, intensity, ventilation and temperature of the inspired air can be adjusted to simulate the sport and environmental conditions in which it is performed if necessary. False negative results for exercise-induced asthma/exercise-induced bronchoconstriction are uncommon.23,24
- Hyperosmolar aerosols of salt and sugar.25,26
- Inhalation of a dry powder preparation of mannitol is now available as a challenge test for airway hyperresponsiveness: the mannitol test provides an alternative to testing by exercise, eucapnic voluntary hyperpnoea and inhaled hypertonic saline in the diagnosis of exercise-induced asthma and exercise-induced bronchoconstriction.26–28

Under- and over-diagnosis

Exercise-induced asthma/exercise-induced bronchoconstriction may be under-diagnosed due to:

- poor perception of symptoms unless the FEV1 falls 20% or more
- post-exercise breathlessness not considered abnormal.

Exercise-induced asthma/exercise-induced bronchoconstriction may be over-diagnosed because:

- people who are overweight and unfit become breathless easily
- cough after exercise commonly occurs in non-asthmatics when they exercise heavily in cool dry air
- winter athletes commonly exhibit cough, breathlessness, wheeze and mucus production29
- a person, usually an athlete, may have vocal cord dysfunction.30

Effect of training

Asthma severity, as reflected by exercise-induced asthma, is not altered by training, but the threshold for respiratory symptoms can increase. This means that after training, the person is likely to:

- have less exercise-induced asthma
- be less breathless
- be less anxious about activity
- feel good
- be less dependent on treatment
- lose less time from school.

Some athletes find warm up prevents them getting exercise-induced asthma during the main game. This beneficial effect may be due to improved delivery of water to the airway surface by the bronchial circulation.

Treatment strategies to manage exercise-induced asthma

Drugs that reduce airway inflammation, inhibit the release of mediators or inhibit the contractile effects of these mediators can be used to manage exercise-induced asthma.35

- Inhaled corticosteroids (ICS): have been shown to significantly reduce the severity of exercise-induced asthma and completely inhibit exercise-induced asthma in 50% of cases following 8–12 weeks treatment.4,9,36,37,11 Sodium cromoglycate, nedocromil sodium or a bronchodilator can be used immediately before exercise (or as rescue medication) until the full effect of ICS is realised. This ICS alone approach is suggested for asthmatics with normal lung function for whom a bronchodilator is not indicated. It is recommended because successful treatment results in 50% of people no longer having exercise-induced asthma and thus the need for pre-exercise medication is avoided.

Practice tips

- Normal values for spirometry should not exclude a diagnosis of exercise-induced asthma/exercise-induced bronchoconstriction.
- Classic symptoms of asthma such as waking at night or early in the morning are not common in athletes who may only have exercise-induced asthma/exercise-induced bronchoconstriction. Objective measurement is recommended before a firm diagnosis is made (especially for those who require permission to inhale a beta2 agonist before a sporting event).4,31
- Exercise-induced asthma/exercise-induced bronchoconstriction is often one of the first signs of asthma32–34 and one of the last to go with treatment.8
- Early identification and treatment of exercise-induced asthma/exercise-induced bronchoconstriction may prevent later development of clinically recognised asthma and changes in lung function.
• **Long-acting beta agonist (LABA)** in combination with ICS: can be used successfully for prevention of exercise-induced asthma in patients with abnormal spirometry and/or more persistent symptoms. The duration of the protective effect of the LABA against exercise induced asthma is reduced when they are taken daily, even in combination with ICS. As a result of this reduction in duration of protection, it would be expected that over time patients will use extra inhalations of SABA before exercise or if breakthrough exercise-induced asthma occurs.

• **Long-acting beta agonist (LABA) alone**: can be used successfully for prevention of exercise-induced asthma. LABAs can be effective for up to 12 hrs when they are used intermittently (less than 3 times a week) but the duration of the protective effect is reduced when these drugs are used daily. Further, recovery time from breakthrough exercise-induced asthma can be prolonged when LABA are used daily.

• **Short-acting beta agonist (SABA)**: provide about 80% protection for up to 2 hrs when given immediately before exercise. SABAs are also effective rescue therapy provided they are not being used to excess. Single-dose LABA or SABA treatment can be beneficial, particularly in young people with normal lung function and no other significant asthma symptoms.

• **Sodium cromoglycate and nedocromil sodium**: are less effective than beta agonists in preventing exercise-induced asthma. They provide 50–60% protection for only 1–2 hours, but have some advantages over beta2 agonists. They do not induce tolerance and can be used several times in a day. The dose can be adjusted and the protective effect is immediate.

• **Leukotriene receptor antagonists**: are also used to control exercise-induced asthma and provide 50–60% protection when given as tablets for up to 24 hrs. They do not induce tolerance and recovery from any residual exercise-induced asthma is rapid, usually occurring within 15 minutes.

### Drug-free strategies

People with exercise-induced asthma may benefit from the following advice:

- Be as fit as possible so that the threshold for exercise-induced asthma is increased (many forms of exercise will not be of sufficient intensity to cause an attack of asthma).
- Exercise in a warm humid environment.
- Do not perform exercise in environment with a high level of allergens (pollen season) or a high level of particulate matter or irritant gases.
- Consider breathing with a mask or through the nose.
- Warm up: this may prevent exercise-induced asthma during the main game.

### Practice points

- ICS treatment for 8–12 weeks reduces severity of exercise-induced asthma and after treatment 50% of people will no longer require medication pre-exercise.
- ICS in combination with a LABA is recommended to prevent exercise-induced asthma for those with abnormal spirometry and persistent symptoms. The duration of the protective effect of LABAs is reduced with daily use (either alone or in combination with ICS). Thus, in the middle of a dosing period, extra doses of the combination or a SABA may be required to protect against exercise-induced asthma.
- Single doses of short- or long-acting beta2 agonist, sodium cromoglycate or nedocromil sodium may significantly inhibit or even prevent exercise-induced asthma when taken immediately before exercise.
- LABAs are best used intermittently to prevent exercise-induced asthma because the duration of their protection against exercise is reduced when they are taken daily, whether alone or in combination with inhaled corticosteroids.
- Leukotriene receptor antagonists reduce severity and duration of exercise-induced asthma. Tolerance does not develop to daily use of these drugs.

---

**Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.**
Use of asthma medications in competitive sport

Many sporting bodies require objective evidence of exercise-induced asthma/exercise-induced bronchoconstriction in order for athletes to use asthma medications during competition.

• The International Olympic Committee now requires documentation of asthma or exercise-induced bronchoconstriction as a prerequisite for permission to use an inhaled beta₂ agonist.³¹

• Regulations regarding the use of certain medications in sport may depend on the different sporting bodies. If a sporting organisation allows the therapeutic use of prohibited substances, the athlete must strictly adhere to the approval procedures. Healthcare providers should advise patients to check with their sport’s relevant sporting organisation.

• The Australian Sports Anti-Doping Authority provides information about Therapeutic Use Exemptions for athletes who suffer from medical conditions requiring treatment with prohibited substances. Go to www.asada.gov.au or call the ASADA tollfree hotline: 1800 020 506 to check the status of substances. The hotline is a confidential service for athletes and their support staff that offers information on the status of Australian pharmaceutical medications and substances in sport.
SUMMARY OF PRACTICE POINTS | LEVEL OF EVIDENCE
--- | ---
Consider the diagnosis of occupational asthma in all new cases of adult-onset asthma, because early diagnosis and avoidance of exposure is associated with the best prognosis. | III
In people with high-risk occupations, the presence of new-onset rhinitis is associated with increased risk for occupational asthma. | III
Consistent improvement in asthma symptoms outside the work environment is a good indicator of occupational asthma. | III
Suspected cases of occupational asthma should be investigated by serial lung function measurements analysed by a validated method. | IV
Serial PEF testing cannot reliably rule out the diagnosis of occupational asthma. | IV
With continued exposure to the causal agent in the workplace, occupational asthma is unlikely to improve and may worsen. | III

Occupational asthma accounts for up to 15% of all adult-onset asthma. It is the most commonly reported occupational respiratory disorder in westernised industrial countries.

Generally, occupational asthma has a poor prognosis and is likely to persist and deteriorate unless identified and managed early and effectively. Where feasible, early referral to a respiratory physician gives patients the best chance of good control or cure. Alternatively, referral to an occupational health physician might be appropriate for some patients. Where specialist services are not readily accessible, GPs can investigate and manage suspected occupational asthma effectively by:

- being aware of high-risk occupations
- taking a very careful history, looking for links between onset of symptoms and occupation-related exposure to potential causal agents
- understanding that the general principles of pharmacotherapy for asthma apply also to occupational asthma. See [Principles of drug therapy](#).

Accurate diagnosis and documentation are essential to support a potential Workers Compensation claim. This would normally require specialist reports. For more information, including details of occupational asthma surveillance schemes in states and territories, contact the National Occupational Health and Safety Commission.

### Definition and mechanism

Occupational asthma is defined as new-onset adult asthma caused by exposure to the workplace environment and not by factors outside the workplace.

Patients with occupational asthma fall into two groups:

1. **Immunological (IgE-mediated)** occupational asthma is characterised by a delay between exposure to a respiratory sensitisier and the development of symptoms.

2. **Non-immunological occupational asthma** typically occurs within a few hours of high-concentration exposure to an irritant at work. This sometimes occurs weeks or months after repeated low-concentration exposure to an irritant.

   - The majority of patients with occupational asthma have immunological asthma.
   - The onset of rhino-conjunctivitis prior to asthma symptoms is more strongly associated with immunological occupational asthma than the non-immunological form.
**Incidence, risk factors and prevention**

**Incidence**

At least 31 out of every 1,000,000 workers in Australia will develop occupational asthma each year. The true incidence is likely to be much higher, because occupational asthma is under-reported.

**Risk factors**

Occupational asthma is most frequently reported among people with the following occupations (not in order of risk):

- animal handlers
- nurses
- bakers and pastry makers
- spray painters
- chemical workers
- timber workers
- food processing workers
- welders.

Cigarette smoking can increase the risk of developing occupational asthma in response to exposure to some sensitising agents.

**Prevention**

Reducing airborne exposure to potential allergens in the workplace lowers workers' risk for becoming sensitised and developing occupational asthma. Respiratory protective equipment is only effective as protection against workplace airborne allergens when worn properly, removed safely, and either replaced or maintained regularly. Such equipment reduces the risk, but does not prevent occupational asthma.

**Diagnosis**

**Practice points**

- Consider the diagnosis of occupational asthma in all new cases of adult-onset asthma, because early diagnosis and avoidance of exposure is associated with the best prognosis. (III)
- In people with high-risk occupations, the presence of new-onset rhinitis is associated with increased risk for occupational asthma. (III)
- Consistent improvement in asthma symptoms outside the work environment is a good indicator of occupational asthma. (III)
- Suspected cases of occupational asthma should be investigated by serial lung function measurements analysed by a validated method. (IV)
- Serial PEF testing cannot reliably rule out the diagnosis of occupational asthma. (IV)

Consider the possibility of an occupational asthma diagnosis in all new cases of adult asthma or rhinitis.

- Ask about exposure to airborne substances at work.
- In addition to taking a standard history, ask about the pattern of symptoms with respect to work (Table 1).

In people whose occupation is associated with a high risk for occupational asthma, the presence of new-onset rhinitis may signal increased risk for developing immunoglobulin E (IgE)-mediated occupational asthma within 12 months.

For patients in high-risk occupations, consider referral to a specialist respiratory physician or occupational physician with expertise in occupational asthma.

For more information on taking a history in a patient with respiratory symptoms, see Diagnosis and classification of asthma in adults and Ongoing care.

**Table 1. Additional questions to ask patients with adult-onset asthma**

Note that these questions are not specific for occupational asthma and may also identify people with asthma due to agents at home, or who experience exercise-induced asthma due to work-related activity.

**Notes**

- What substances do you handle at work? Are there any dusts, powders or substances in the air?
  - Several hundred agents have been reported to cause occupational asthma and new causes are reported regularly in medical literature.

- When did the symptoms start? Have there been any changes at work?
  - Consider with respect to the timing of any changes in the patient's work environment or lifestyle.

- Are symptoms different when not at work? Do symptoms improve on days off?
  - Any improvement in symptoms when away from work is a good indicator of occupational asthma.

- Do symptoms improve during a long holiday from work?
  - Regular improvement of symptoms when away from work is a more reliable indicator of occupational asthma than increase in symptoms when at work.

- Does anyone else at work have a similar problem?
  - Indicates that the workforce and workplace should be assessed.

A diagnosis may not be possible once exposure has ceased, so a specialist referral should be arranged first if possible.
Objective tests
Lung function tests and blood testing may help establish the diagnosis of occupational asthma.

Peak expiratory flow
Serial measurement of peak expiratory flow (PEF) is a useful test for occupational asthma.5–10
• Measure PEF at least four times a day over at least three weeks, at and away from work, and analyse the results by a validated method. A UK group of researchers has developed a computer program (OASYS) as a validated tool to assist in diagnosing occupational asthma from serial PEF records (See www.occupationalasthma.com).11
• Serial PEF cannot reliably rule out the diagnosis of occupational asthma. As a diagnostic test for occupational asthma, serial PEF (when performed and interpreted according to established protocols) is associated with a low rate of false positives, but approximately 30% false negatives.

Practice tip
Peak expiratory flow has a special role in the initial investigation of occupational asthma, where the frequent lung function testing required would make spirometry impractical.

The gold standard for diagnosis of occupational asthma is a specific challenge with the suspected agent. However, such challenges are only done in specialist referral centres.

Specific immunoglobulin testing
Blood tests or skin prick tests for specific immunoglobulin E (IgE) for suspected allergens can help to identify the causal agent, when interpreted together with evidence from:
• a detailed history about symptoms
• the temporal relationship of symptoms to the workplace
• exposure to substances at work.

Where possible, the diagnosis of occupational asthma should be confirmed by a specialist in this field. Documentation of specialist confirmation will support a potential claim for Workers Compensation. For information about access to referral, contact the Thoracic Society of Australia and New Zealand.

Prognosis

Practice point
With continued exposure to the causal agent in the workplace, occupational asthma is unlikely to improve and may worsen.12 (III)

• If the diagnosis of occupational asthma is made early and the person avoids further exposure to the respiratory sensitiser/s in the workplace, symptoms may resolve completely.1
• Improvement is greatest in patients with near-normal lung function at the time of diagnosis and a short duration of symptoms (<12 months).1
• With continued exposure to the workplace sensitiser, occupational asthma is unlikely to improve and may worsen.1
• Symptoms and functional impairment may persist for many years after avoidance of further exposure to the causative agent.1
• Death due to occupational asthma has occasionally been reported.13

Management of occupational asthma
• Ideal management includes complete and permanent avoidance of exposure to the environmental agents causing asthma. However, this is not always possible for individual patients.
• If complete avoidance of the allergen is not possible, the worker should be moved to an area of the workplace with lower exposure or occasional exposure and remain under regular specialist medical surveillance.
• Medical management should be as for other patients with asthma. See Principles of drug therapy and Ongoing care
• If the patient’s employer has access to an occupational health service, the general practice can liaise with this service with the patient’s consent.

Further information
British Occupational Health Research Foundation
(www.bohrf.org.uk)
OASYS and Occupational Asthma
(www.occupationalasthma.com)
Pregnancy and asthma

Maintaining good asthma control during pregnancy is important for the health of both the mother and baby. Appropriate and ongoing asthma care can successfully manage deteriorations and exacerbations of asthma in most cases. At all times during pregnancy, the use of any medicine is a balance between the justification for its use in maintaining asthma control and the potential for adverse effects.

Breathlessness during pregnancy is common and is usually due to hormonal changes, not asthma. Spirometry can assist in determining the cause. The use of bronchial provocation tests for the diagnosis of asthma in pregnant or lactating women should only be performed on the advice of a respiratory specialist.

Before pregnancy

Women with asthma who are planning to become pregnant should stop smoking.

Women should be advised that, if they become pregnant:
• their asthma and pregnancy may interact
• good asthma control is important
• their asthma should be reviewed regularly
• many asthma medications, including most inhaled corticosteroids (ICS), have a good safety profile and should be continued during pregnancy.

Because of the risk of precipitating an exacerbation, asthma medications should not be changed in women whose asthma is well controlled and who present after they have become pregnant. However, in women who are planning a pregnancy and already using ICS should switch to budesonide, a Category A drug. More data on use in pregnant women are available for budesonide than for other ICS. However, there are no data indicating that other ICS are unsafe during pregnancy.

Review asthma control after any change in the medication regimen.

Practice tips

Women with asthma who are planning to become pregnant should be advised to stop smoking.

Women should be advised that, if they become pregnant:
• their asthma and pregnancy may interact
• good asthma control is important
• their asthma should be reviewed regularly
• many asthma medications, including most ICS, have a good safety profile and should be continued during pregnancy.
Antenatal care

Exacerbations of asthma requiring medical intervention occur in about 20% of pregnant women and about 6% will require hospitalisation. Regular evaluation and monitoring of asthma control is, therefore, recommended throughout pregnancy. Poorly controlled asthma increases the risk of pre-eclampsia, preterm birth, low birth weight and perinatal mortality. Good asthma control can reduce these risks.

The goals of management during pregnancy are to maintain asthma control so as to ensure the oxygen supply required for normal foetal development, as well as to maintain maternal health and quality of life. The pharmacological treatment of asthma during pregnancy should be the same as for non-pregnant women. (See Medication during pregnancy and lactation)

- Doses of ICS should be the minimum necessary to control symptoms and maintain normal or best lung function.
- Peak expiratory flow monitoring and regular review of asthma every 4–6 weeks is recommended. This can provide reassurance for the pregnant woman and her healthcare providers. Close cooperation between all health professionals caring for the pregnant patient is important to ensure the best asthma management.
- Acute asthma exacerbations may reduce the amount of oxygen available to the foetus. Any deterioration in symptoms should be managed promptly.
- Trigger factors should be avoided or minimised where possible. Minimise exposure to known allergens and irritants.

Practice points

- Poorly controlled asthma increases the risk of pre-eclampsia, prematurity, low birth weight and perinatal mortality. (III) Good asthma control reduces these risks. (IV)
- The pharmacological treatment of asthma during pregnancy should be the same as in non-pregnant women. (IV)
- Women who are planning a pregnancy and already using ICS should switch to budesonide, a Category A drug. (IV)

Asthma exacerbations during pregnancy

Asthma exacerbations during pregnancy may be related to poor pregnancy outcomes. Exacerbations during pregnancy:

- occur primarily between 17 and 36 weeks gestation
- are often triggered by viral infection and non-adherence to ICS medication
- significantly increase the risk of having a low-birth-weight baby.

The effective management and prevention of asthma exacerbations during pregnancy is important for the health of both mother and baby.

- ICS use may reduce the risk of exacerbations during pregnancy.
- Prescribe oral corticosteroids in pregnant women when clinically indicated.

Practice tips

Asthma exacerbations during pregnancy should be managed:

- promptly
- in the same way as an exacerbation at any other time
- with oral corticosteroids if clinically indicated.

If antibiotics are to be used their safety in pregnancy should be confirmed (refer to ADEC. Prescribing medicines in pregnancy. 4th edition, 1999.).

Delivery

Except in the most severe cases, asthma should not preclude a vaginal delivery. Caesarean section should be no more common than in women without asthma. Exacerbations of asthma are uncommon during labour and delivery.

Post-partum phase

Review asthma regularly after delivery.

Remind parents that passive smoking increases the risk of childhood asthma and other respiratory conditions in their child. The link between exposure to environmental tobacco smoke in early childhood and increased risk of respiratory illnesses, including asthma, has been well documented in epidemiological studies. Avoidance of environmental tobacco smoke may reduce the risk of childhood asthma.
Breastfeeding should be encouraged as it may reduce the risk of childhood asthma, especially in children with a family history of atopy. For more information on breastfeeding and asthma, see Prevention of asthma.

Practice tips
- Advise pregnant women to avoid passive smoking.
- Encourage breastfeeding.

Medications during pregnancy and lactation
It is safer for pregnant women to maintain control of their asthma with appropriate medications than for them to have asthma symptoms and exacerbations.1
- The pharmacological treatment of asthma during pregnancy should be the same as in the non-pregnant state. If oral corticosteroids are clinically indicated for an exacerbation they should not be withheld because a woman is pregnant.1
- If maintenance treatment with ICS was necessary before the pregnancy, it should be continued during pregnancy.1 As in the non-pregnant state, the dose should be the minimum necessary to control symptoms and maintain normal or best lung function.
- Most medications for asthma have good safety profiles in pregnancy. There is some evidence that the use of oral corticosteroids (particularly in the first trimester) is associated with a slight increase in the incidence of cleft lip with or without cleft palate. This finding is based on small numbers and from studies not specifically designed to assess the risk.
- There are limited data describing the effectiveness and/or safety of using combination therapy during pregnancy.1 To date, there are no studies examining the effects of long-acting beta2 agonists on pregnancy outcomes.6 Salmeterol and eformoterol are rated Category B3 drugs. These medicines should not be withdrawn in women who present after they have become pregnant if they are controlling symptoms. It is recommended that these agents be avoided, if possible, during the first trimester. No safety studies of leukotriene receptor antagonists in pregnancy have been published. They should only be used where other medications have not achieved satisfactory asthma control.
- Asthma medications can enter breast milk, but their concentrations are generally so small that they appear to have no adverse effects on the baby.3


Practice points
- Most medications for asthma have good safety profiles in pregnant women. (III)
- If oral corticosteroids are clinically indicated for an exacerbation they should not be withheld because a woman is pregnant. (IV)
- The ICS regimen should not be changed in pregnant women whose asthma is well controlled. However, women who are planning a pregnancy and already using inhaled corticosteroids should switch to budesonide, a Category A drug. (IV)
- Salmeterol and eformoterol should not be stopped if a pregnant woman is already using them, but if possible, they should be avoided during the first trimester.

Smoking
Women should stop smoking before becoming pregnant and avoid environmental tobacco smoke during pregnancy. Parents should ensure their children are not exposed to tobacco smoke.
- Smoking during pregnancy increases the risk of premature labour, low birth weight and respiratory disorders during early infancy.
- Children exposed to tobacco smoke during gestation or infancy are more likely to develop asthma.

Practice points
Remind parents that passive smoking increases the risk of childhood asthma and other respiratory conditions in their child. Avoidance of environmental tobacco smoke may reduce the risk of childhood asthma. (III-2)

The Asthma Foundation of NSW has useful resources on asthma and pregnancy: Healthy pregnancy in women with asthma, an information paper for health professionals, 2006, and a consumer brochure, Asthma and healthy pregnancy (available at: www.asthmansw.org.au).
Asthma in the elderly

Asthma in elderly patients is more common than previously understood.1–5

- The lifetime asthma prevalence among middle-aged and older Australians is approximately 15%.4,6 Asthma prevalence in the general adult population is estimated at approximately 10–12%.7,8
- Emerging international evidence suggests that the prevalences of both asthma and chronic obstructive pulmonary disease (COPD) are increasing.9–11

The risk of dying from asthma increases with age. The majority of asthma deaths occur in people aged 65 and over, particularly during the winter months.7

Asthma in elderly patients is under-diagnosed

It has been estimated that up to one-third of elderly people with asthma are not identified by their doctors.5,12
- Lack of awareness of the possibility of new-onset asthma in the elderly may be a factor in both under-reporting and misdiagnosis.13
- Patients and doctors often attribute respiratory symptoms to ageing or common diseases of the elderly.14,15
- Elderly people may be unaware of reduced respiratory function when activities of daily living are limited by other conditions, or when perception of breathlessness is reduced.16
- Comorbidity may make the diagnosis of asthma in older people more difficult.

Identifying asthma in elderly patients is clinically important:
- Asthma tends to be more severe in older patients than younger adults, based on spirometric lung function parameters, clinical features of asthma (emergency visits and hospitalisation rates) and comorbidities.5,12,17–21
- Mortality rates are higher in elderly patients than in younger age groups, and acute asthma attacks more rapidly fatal.22

SUMMARY OF PRACTICE POINTS

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Always attempt to make the distinction between asthma and COPD, or determine that both are present, so that the optimal treatment can be prescribed.</td>
<td>✓</td>
</tr>
<tr>
<td>Spirometry is mandatory for detecting airflow limitation in both asthma and COPD</td>
<td>✓</td>
</tr>
<tr>
<td>The possibilities of both asthma and COPD must be considered in all patients with cough or unexplained breathing difficulty during physical activity.</td>
<td>✓</td>
</tr>
<tr>
<td>Demonstration of a small degree of acute reversibility to bronchodilators alone does not distinguish asthma from COPD.</td>
<td>✓</td>
</tr>
<tr>
<td>Avoid the use of oral corticosteroids in treatment trials in elderly patients.</td>
<td>✓</td>
</tr>
<tr>
<td>Warn patients that delay of effective treatment during an acute episode through over-reliance on nebulisers increases the risk of life-threatening asthma.</td>
<td>✓</td>
</tr>
<tr>
<td>Be aware that perception of airflow limitation is reduced in older people. Always ask “Can you feel any difference after the reliever?” before measuring post-bronchodilator FEV₁.</td>
<td>✓</td>
</tr>
<tr>
<td>Check inhaler technique and adherence whenever asthma is reviewed.</td>
<td>✓</td>
</tr>
<tr>
<td>Set up an effective recall process to ensure annual influenza re-vaccination and review of pneumococcal vaccination status in all elderly patients with asthma, even in those with mild asthma.</td>
<td>✓</td>
</tr>
<tr>
<td>Consider a PEF-based asthma action plan for patients who have shown poor perception of airflow limitation.</td>
<td>✓</td>
</tr>
</tbody>
</table>
Asthma in the elderly

• Asthma is associated with significant disability, depression and impairment of mobility in older patients.1,12,17,18

• In the general population, long-term delay in the diagnosis of respiratory symptoms can lead to progressive and irreversible loss of pulmonary function,23 while the benefits of prompt treatment are clear, even for mild asthma.24–27 Similar benefits may be expected in the elderly.

Diagnosis of asthma in older patients

As for younger adults, the diagnosis of asthma in older patients is based on:

• history
• physical examination
• supportive diagnostic testing (e.g. spirometry).

For more information, see Diagnosis and classification of asthma in adults.

Diagnostic difficulties in the elderly are listed in Table 1. Despite these difficulties, always attempt to make the distinction between asthma and COPD, because they have different natural histories and expected response to therapy.28

There are many asthma phenotypes, and no single item or procedure can definitively determine the presence of asthma. Diagnosis involves an overall assessment of the patient’s medical history, physical examination, laboratory test results and observation over time.

Spirometry is the most effective diagnostic tool available to assist general practitioners in the accurate diagnosis of asthma. Spirometry is mandatory for detecting airflow limitation in both asthma and COPD,29,30 and helps distinguish between these diseases.

For more information, see COPD and asthma.

Suggested diagnostic steps in the elderly

A useful diagnostic approach in elderly patients with suspected respiratory disease is:

1. Aim to identify all patients with airflow limitation (either COPD or asthma)
2. Exclude other conditions
3. Distinguish COPD from asthma
4. Consider the possibility that asthma and COPD are both present and overlap.

Practice tip

A useful diagnostic approach in elderly patients with suspected respiratory disease is:

first: aim to identify all patients with airflow limitation (either COPD or asthma)
second: exclude other conditions
third: distinguish COPD from asthma and determine whether one or both are present.

Identifying patients with airflow limitation

Helpful questions to ask when taking a history in older adults with suspected airflow limitation (asthma, COPD or both) are listed in Table 2.

Consider symptoms, smoking history and allergies when taking the history.

Symptoms

The possibilities of asthma and COPD must both be considered in all patients with cough.

• In adults up to 75 years presenting with cough, around half may have asthma or COPD.32,33

• In patients aged 65 and over with new-onset asthma, the symptoms most frequently experienced are cough, wheeze and dyspnoea. Onset of symptoms commonly coincides with upper respiratory tract infection.34

Table 1. Diagnostic difficulties in the elderly

<table>
<thead>
<tr>
<th>Difficulty</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complex differential diagnosis – symptoms of asthma are non-specific and overlap with other conditions that are relatively common among older adults, including COPD, heart failure and lung cancer (e.g. it may be very difficult to distinguish between asthma and COPD in a smoker with a history of asthma).</td>
<td></td>
</tr>
<tr>
<td>Poor recognition of symptoms – physiological changes of ageing can result in reduced perception of airflow limitation by patient.16</td>
<td></td>
</tr>
<tr>
<td>Limited utility of direct tests of airway hyperresponsiveness – these are much less sensitive in people aged over 50 years than in younger adults.31</td>
<td></td>
</tr>
<tr>
<td>Comorbidity – deafness, frailty, physical deconditioning and the presence of other chronic conditions (e.g. arthritis, heart disease) can contribute to problems in interpreting symptoms and performing lung function tests.</td>
<td></td>
</tr>
<tr>
<td>Cognitive impairment may be a barrier to obtaining a clear and thorough history.</td>
<td></td>
</tr>
<tr>
<td>Social isolation may delay presentation to medical services.</td>
<td></td>
</tr>
</tbody>
</table>
Smoking history
Smoking history should be taken in all older patients with suspected respiratory disease. Smoking history is a major risk factor for COPD, but cannot rule out the diagnosis of asthma, especially in cases that overlap.

Allergy
History should include questions about history of allergy (hay fever, eczema).
- Atopy has been identified as an important predictor of asthma in the elderly as well as in other age groups.\textsuperscript{12,35,36}
- Ask about previous history of allergies and about seasonal response to environmental, household or animal allergens.\textsuperscript{12}
- However, the absence of atopy or other immunological markers of asthma does not rule out an asthma diagnosis. Asthma may be triggered more often by respiratory tract viruses than allergies in older people.\textsuperscript{34}
- Ask about family history as well as past history and of respiratory symptoms. Older people with current asthma symptoms commonly have a family history of asthma and childhood respiratory problems.\textsuperscript{36}

For more information about allergic rhinitis, see Asthma and Allergy.

Table 2. Useful questions for identifying airflow limitation in older adults

<table>
<thead>
<tr>
<th>Diagnostic questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you had any wheezing or whistling in your chest in the last 12 months?</td>
</tr>
<tr>
<td>Have you had this wheezing whistling when you did not have a cold?</td>
</tr>
<tr>
<td>Have you woken at night with feeling of tightness in chest in the last 12 months?</td>
</tr>
<tr>
<td>Have you been woken by an attack of shortness of breath in the last 12 months?</td>
</tr>
<tr>
<td>Have you been woken by an attack of coughing in the last twelve months?</td>
</tr>
<tr>
<td>Have you had an attack of asthma in the last 12 months?</td>
</tr>
<tr>
<td>Are you currently taking any medicine for asthma?</td>
</tr>
<tr>
<td>Do you have any nasal allergies including hay fever?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Are you a smoker or ex-smoker?</td>
</tr>
<tr>
<td>Did you have any allergies as a child?</td>
</tr>
<tr>
<td>What type of work have you done for most of your life?</td>
</tr>
</tbody>
</table>

Excluding diagnoses other than asthma and COPD
A differential diagnosis for respiratory symptoms in older adults is set out in Table 3.

Where there in any uncertainty as to the cause of new symptoms, a chest X-ray should be done to rule out other significant morbidity or complications (eg pneumothorax) or other diagnoses (e.g. congestive heart failure or lung carcinoma).\textsuperscript{35,37,38}

Table 3. Important causes for respiratory symptoms in the elderly

<table>
<thead>
<tr>
<th>Pulmonary</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td></td>
<td>Bronchiectasis</td>
</tr>
<tr>
<td></td>
<td>Pulmonary emboli</td>
</tr>
<tr>
<td></td>
<td>Pulmonary fibrosis</td>
</tr>
<tr>
<td></td>
<td>Pneumonia, bronchitis</td>
</tr>
<tr>
<td></td>
<td>Pleural effusion</td>
</tr>
<tr>
<td></td>
<td>Pneumothorax</td>
</tr>
<tr>
<td></td>
<td>Malignant disease</td>
</tr>
<tr>
<td></td>
<td>Aspiration pneumonitis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiac</th>
<th>Arrhythmias</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ischaemic heart disease</td>
</tr>
<tr>
<td></td>
<td>Valvular heart disease</td>
</tr>
<tr>
<td></td>
<td>Cardiomyopathies</td>
</tr>
<tr>
<td></td>
<td>Pericardial disease</td>
</tr>
</tbody>
</table>

| Neurological    | Respiratory muscle weakness |
|-----------------| Motor neurone disease |
|                 | Myasthenia gravis |

| Drug-related    | Oral and topical beta\textsubscript{2} adrenergic receptor antagonists |
|-----------------| Nonsteroidal anti-inflammatory drugs |
|                 | Angiotensin converting enzyme (ACE) inhibitors |

| Other           | Vocal cord dysfunction |
|-----------------| Gastroesophageal reflux disease |
|                 | Anaemia |
|                 | Anxiety |
|                 | Tracheal stenosis |

Adapted from Dow L. 1998\textsuperscript{14}
Asthma in the elderly is often difficult to distinguish from COPD. Clinical signs of asthma and COPD overlap. Discriminating between asthma and COPD (Table 4), or concluding that both diseases are present and overlapping, is based on the following:

- symptom information
- degree of reversibility of airflow limitation
- peak expiratory flow (PEF) variability
- bronchial hyperresponsiveness
- history of allergy.

Note that:
- single symptoms do not discriminate between asthma and COPD.39

Maximal relevant diagnostic information is obtained when spirometry is performed before and after bronchodilator and before and after a treatment trial. The diagnosis might be made with confidence only after observing and collecting clinical data for weeks or months.

For more information, see COPD and asthma.

### Spirometry in the elderly

As in younger adults, spirometry findings must be interpreted carefully, with reference to clinical findings. Clinicians should be aware of potential pitfalls in the interpretation of spirometry in the elderly, listed in Table 5.

- The diagnosis of asthma can be made with a high degree of confidence when post-bronchodilator FEV1/FVC ratio is greater than 70% and acute reversibility is demonstrated after administration of bronchodilator (a postbronchodilator increase of ≥ 200 ml and ≥ 12% in FEV1 or FVC).
- The diagnosis of probable asthma can be made by demonstrating acute reversibility after bronchodilator, even when post-bronchodilator FEV1/FVC ratio is less than 70%.
- Reversibility of airflow limitation after a therapeutic trial helps confirm the diagnosis of asthma.

For more information on criteria for reversibility of airflow limitation, see Diagnosis and classification of asthma in adults.

### Table 4. Factors that distinguish asthma from COPD

<table>
<thead>
<tr>
<th>Factor Present In</th>
<th>Asthma</th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young age at onset</td>
<td>Often</td>
<td>Almost never</td>
</tr>
<tr>
<td>Sudden onset of disease</td>
<td>Often</td>
<td>Almost never</td>
</tr>
<tr>
<td>Smoking history</td>
<td>Sometimes</td>
<td>Almost always</td>
</tr>
<tr>
<td>Allergy</td>
<td>Often</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>Often</td>
<td>Often</td>
</tr>
<tr>
<td>Wheezing</td>
<td>Often</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Coughing</td>
<td>Sometimes</td>
<td>Often</td>
</tr>
<tr>
<td>Sputum production</td>
<td>Seldom</td>
<td>Often</td>
</tr>
<tr>
<td>Chronic airflow limitation</td>
<td>Seldom</td>
<td>Almost always</td>
</tr>
<tr>
<td>Variable airflow limitation</td>
<td>Almost always</td>
<td>Seldom</td>
</tr>
<tr>
<td>Reversible airflow limitation</td>
<td>Almost always</td>
<td>Almost never</td>
</tr>
<tr>
<td>Airway hyperresponsiveness</td>
<td>Almost always</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Diurnal peak expiratory flow variability</td>
<td>Almost always</td>
<td>Sometimes</td>
</tr>
</tbody>
</table>

Adapted from Van Schayck C, 199640

These factors can help distinguish between asthma and COPD, but be aware that both conditions may be present.

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
COPD

- Spirometry is mandatory for the detection of early stages of COPD in general practice, and at least doubles the proportion of patients identified.\(^{30}\)
- The diagnosis of COPD is based on a demonstration of airflow limitation (post-bronchodilator FEV\(_1\)/FVC ratio of less than 70%), together with lack of acute reversibility after administration of bronchodilator. This criterion may result in false positives in older people,\(^{41}\) but that is preferable to under-diagnosis, given the potential poor health outcomes in older people.
- Lack of full reversibility after a therapeutic trial helps confirm the diagnosis.

See COPD and asthma.

Overlap of asthma and COPD

The presence of overlapping COPD and asthma is a strong possibility in a patient whose clinical profile includes all of the following features:

- Age over 45 years
- A history of smoking
- Reversibility of airflow limitation acutely after bronchodilator or over time
- Post-bronchodilator FEV\(_1\)/FVC < 70%.

Managing asthma in elderly patients

Drug treatment

As for patients of all age groups:

- the choice of initial treatment is guided by the severity of untreated asthma at the time of diagnosis.
- subsequent modifications of the treatment regimen will depend on the degree of symptom control achieved at regular ongoing review.
- the diagnosis should be reconsidered in a patient whose symptoms respond poorly to therapy.\(^{49}\)

Factors affecting the choice of delivery device in older patients

Comorbidities in older patients will influence the choice of delivery devices:

- Patients who are frail, weak, or have arthritis affecting the hands may need to use additional aids or undergo a trial of various devices to determine the optimal delivery method.
- Patients with cognitive disorders may require a carer to help them use MDIs and spacers.
- Delivery of drugs by nebuliser may be necessary in some patients.
Prescribing issues
When prescribing asthma medications for elderly patients, choose doses cautiously and monitor closely for adverse effects. Clinical trials conducted for registration purposes have generally included few elderly patients.

Consider these issues and consult the Approved Product Information as necessary:
- When prescribing oral corticosteroids, consider the possibility of reactivation of tuberculosis and monitor closely, particularly in those born in countries with high prevalences.
- Lower initial doses (compared with general adult doses) are recommended for some drugs (e.g., salbutamol).
- Clearance of some drugs (e.g., theophylline) is decreased in the elderly and in those with impaired liver function.
- Consider potential interactions with other drugs, e.g.:
  - The risk of hypokalaemia is increased by the concomitant use of beta-agonists and diuretics
  - Theophylline and aminophylline interact with a range of agents. If these are used, start with a low dose and monitor closely for drug–drug interactions.
- Elderly patients with multiple comorbidities may experience difficulties taking complex medication regimens correctly. A Home Medicines Review may be useful. For more information, see The role of the community pharmacist.

Asthma and diabetes
The early use of high-dose inhaled corticosteroids in response to potential exacerbations might be considered in patients with diabetes, in order to avoid the use of oral corticosteroids.

Patients with diabetes need to understand how to control hyperglycaemia, should it be necessary to initiate a short course of oral prednisolone during an asthma exacerbation.

Patient education
As for other age groups, offer patients and carers self-management education, and not only information. This education should be aimed to help them integrate previous ideas and beliefs about asthma with current knowledge. For more information, see Provide self-management education.

- Cognitive status, dexterity and eyesight must be taken into account when educating patients about the roles and correct use of medicines, and use of inhalation devices (Table 6).
- Ensure that patients and carers are given clear information on when to call emergency services. Inappropriate reliance on nebulisers may delay effective treatment.

Practice points
- Avoid the use of oral corticosteroids in treatment trials in elderly patients.
- Warn patients that delay of effective treatment during an acute episode through over-reliance on nebulisers increases the risk of life-threatening asthma.
- Be aware that perception of airflow limitation is reduced in older people. Always ask “Can you feel any difference after the reliever?” before measuring post-bronchodilator FEV1.
- Check inhaler technique and adherence whenever asthma is reviewed.

Table 6. Patient-related factors to consider when choosing a delivery system

<table>
<thead>
<tr>
<th>Aspects to consider</th>
<th>Delivery system notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Strength to operate the device</td>
<td>• MDI versus breath-activated devices</td>
</tr>
<tr>
<td>• Inspiratory flow rate</td>
<td>• Spacers</td>
</tr>
<tr>
<td>• Coordination</td>
<td></td>
</tr>
<tr>
<td>• Agility</td>
<td></td>
</tr>
<tr>
<td>Visual acuity and ability to judge fill status</td>
<td>• Dose counters require good eyesight</td>
</tr>
<tr>
<td>Strength and function of hands</td>
<td></td>
</tr>
<tr>
<td>• Cognitive status</td>
<td>• Haleraids for patients with arthritis or disability</td>
</tr>
<tr>
<td>• Understanding of the roles of each medication</td>
<td>• Where possible, don’t mix inhaler types within a treatment regimen</td>
</tr>
<tr>
<td>• Where possible, don’t mix inhaler types within a treatment regimen</td>
<td></td>
</tr>
<tr>
<td>• Reinforce when each medication should be taken</td>
<td>• Reinforce role of nebulisers</td>
</tr>
</tbody>
</table>

Review of asthma in the elderly patient

Practice points
- Set up an effective recall process to ensure annual influenza re-vaccination and review of pneumococcal vaccination status in all elderly patients with asthma, even in those with mild asthma.
- Consider a PEF-based asthma action plan for patients who have shown poor perception of airflow limitation.
The principles of asthma review in the elderly patient are the same as for other age-groups with asthma. The appropriate frequency of review depends on severity e.g., weekly review may be appropriate in a patient with a recent life-threatening asthma attack; review every 6 or 12 months may be suitable for someone with stable mild persistent asthma.

- Ask whether the patient has experienced any problems with asthma, medication or monitoring.
- Ask about night waking with asthma, morning symptoms, asthma-related limit of normal activity, shortness of breath, wheeze and short-acting beta-agonist (SABA) use. For questionnaires available for use in monitoring asthma, see Ongoing care.
- Ask whether there have been any changes in the use of medications.
- Ask about adherence to the treatment plan.
- Ask about any changes in the environment.
- Ask whether there are any aggravating factors (e.g. gastric reflux).
- Ask whether the person took his or her reliever bronchodilator medication.
- Perform spirometry before and after bronchodilator, and check perception of post-bronchodilator effect.
- Check device technique.
- Check the patient’s (and/or carer’s) understanding of the asthma action plan by asking “What would you do if …?”.

Factors that commonly complicate the monitoring of asthma control in older people include:

- reduced perception of airflow limitation
- comorbidities (e.g. poor eyesight, hearing impairment, weakness due to osteoarthritis, cognitive deficits, neurological deficits secondary to cerebrovascular disease)
- psychosocial issues (e.g. lack of carer, dependence, lack of confidence, depression, perceived and actual financial barriers, resistance to accepting the diagnosis, low motivation).

Consider these strategies to overcome common difficulties:

- For patients with impaired grip strength, add a device to a standard inhaler to make actuation easier (e.g. Haleraid).
- Put a large, easily visible marker on the PEF meter to make it easier for a person with poor eyesight to judge PEF relative to best recorded value (compared with reading from the scale).

- For patients who cannot perform PEF monitoring (e.g. due to stroke or dementia), teach a carer to observe for signs that indicate increased respiratory work:
  - inability to speak in sentences of more than a few words between breaths
  - respiratory distress or marked anxiety
  - acute cognitive impairment compared with usual status.

### Perception of airflow limitation

Assessment of the patient’s ability to perceive changes in airway function is an important part of the assessment of older patients, both when initiating treatment and at each subsequent review.

Perception of airway function can be assessed in two ways:

1. Asking about the use of SABAs. An elderly patient who gives a history of using SABAs in response to asthma symptoms probably has reasonably good perception of airflow limitation.

2. Checking whether the person is aware of a change in symptoms or ease of breathing when a large post-bronchodilator response in FEV₁ (or PEF) is measured.

   - Routinely ask “Can you feel any difference after the reliever?” before measuring post-bronchodilator FEV₁. If other staff (e.g. practice nurses) are performing spirometry, train them to include this question with all older patients.

   - If the patient has not perceived any change despite a large response (e.g. an increase in FEV₁ of > 20% and > 400 mLs), it is advisable to write a PEF-based asthma action plan.

### Practice tip

Schedule a consultation specifically to assess asthma if possible, because:

- It is difficult to check all these aspects of asthma at one review
- Asthma control can only be assessed when exacerbations are absent.

### Vaccination

Check the status of influenza and pneumococcal vaccination every year in February–March. Ensure that an effective recall process is in place to ensure annual influenza re-vaccination in all elderly patients with asthma – even in those with mild asthma – and that initial
pneumococcal vaccination and subsequent revaccination occurs in line with NHMRC recommendations.

**Acute exacerbations and action plans in elderly patients**

Comorbidity must be considered when planning management of asthma exacerbations in the elderly.

- Where feasible, a PEF-based asthma action plan should be considered for patients who have shown poor perception of airflow limitation.
- A large-print (or handwritten) action plan, or an audiotape of the action plan may benefit visually impaired patients.
- Patients whose first language is not English may need an audiotape of the action plan in their native language.
Other comorbidities

Obstructive sleep apnoea syndrome (OSA) is the most common organic sleep disorder, and is almost as common as asthma. It is characterised by habitual snoring and repetitive obstructive breathing events during sleep, leading to sleep fragmentation, hypoxemia and poor daytime functioning.

Health professionals working with people with asthma should try to identify OSA, because of its potential adverse effect on asthma. Snoring and OSA may worsen sleep disturbance in asthma, while effective treatment for OSA might improve nocturnal asthma symptoms that are otherwise difficult to control.

OSA in adults

- The prevalence of symptomatic adult OSA is approximately 2% in women and 4% in men. Obesity and male sex are the major risk factors. The prevalence of OSA is also higher in late middle age, and in those with asthma, acromegaly, arterial hypertension and heart disease, or type 2 diabetes.
- Snoring and witnessed apnoeas are common among adults with asthma or rhinitis.
- Untreated OSA can cause excessive sleepiness and impaired cognition, and might also contribute to premature cardiovascular disease.

OSA in children

- The prevalence of OSA in children is only slightly lower than in adults. In children, OSA is often caused by adenotonsillar hypertrophy.
- Snoring and, possibly, obstructive apnoeas and hypopnoeas are relatively common in children with a history of wheezing or asthma.
- Untreated OSA has been associated with learning and behavioural difficulties in children.

Aetiology of OSA

The precise mechanism of OSA in adults is not well understood. Factors that can contribute to the...
The development of upper airway obstruction in sleep include anatomical, mechanical, neurological, and inflammatory changes in the pharynx, as well as abnormalities of central respiratory control.  

**Concurrence of snoring, OSA, wheeze and asthma**

It is not known for certain whether there is a causal association between snoring/OSA and wheeze/asthma, or the direction of causality.

- In children with asthma and allergic rhinitis, increased upper airway resistance 12,13,17 or lymphoid hypertrophy18 might promote snoring and upper airway obstruction during sleep.
  
  For more information on allergic rhinitis, see Asthma and Allergy.

- Oxidative stress associated with recurrent obstructive events and nocturnal hypoxemia in OSA might worsen or exacerbate asthma by raising levels of circulating inflammatory mediators (e.g. tumour necrosis factor, endothelin, vascular endothelial growth factor, interleukin 6) that have been associated with airway pathology of asthma.19

- Habitual snoring was an independent predictor of new-onset wheeze and night-time asthma symptoms over 5–10 years of wheeze and night-time asthma symptoms in a large cohort study.20

- A very high prevalence of OSA has been reported among patients with unstable asthma receiving long-term or frequent intermittent oral corticosteroid therapy, possibly secondary to weight gain.21

- Bronchial hyperreactivity has been reported to be more common in patients with OSA than in those with snoring but not OSA, and may respond to continuous positive airway pressure (CPAP) therapy.22

- Upper airway narrowing occurring during sleep, with or without snoring and obstructive sleep apnoea, has been suggested as a mechanism for nocturnal asthma worsening.23 One hypothesis is that a subgroup of asthmatic patients with small pharynxes may have enhanced vagal stimulation during sleep, compared with other asthmatic patients.24 This is supported by data from an animal study suggesting that upper airway vibration, as occurs in snoring, may increase airway smooth muscle tone via a parasympathetic neural reflex.25

**Treatment**

Continuous positive airway pressure is the gold standard treatment for OSA.

- Small uncontrolled studies in adults with asthma and OSA have reported an increase in daytime and nighttime peak expiratory flow26 and reduced nocturnal asthma symptoms26,27 following CPAP treatment, compared with baseline.

- Improvements in nocturnal symptoms have been reported after 2 months’ CPAP therapy in patients who had previously experienced persistent nocturnal asthma despite optimal drug treatment.27

**Clinical implications of OSA in asthma management**

- Snoring and obstructive sleep apnoea may worsen sleep disturbance in patients with asthma.

- Practitioners treating children with asthma should be aware that habitual snoring and obstructive sleep apnoea are more common in these children.

- Regardless of its relationship to asthma, OSA may require investigation and therapy because of its detrimental effects on function and quality of life.

- In patients with asthma symptoms that are refractory to standard drug treatments, treatment for OSA may improve asthma symptoms, particularly nocturnal symptoms.

**Gastro-oesophageal reflux and obesity**

**Gastro-oesophageal reflux (GORD)**

**Practice point**

A trial of acid suppression therapy may be worthwhile if GORD is suspected but treatment for reflux does not predictably improve asthma control. (I)

GORD and OSA commonly co-occur.28

- Asthma appears to be more prevalent among people with GORD.20,28 The occurrence of GORD after bedtime is strongly associated with both asthma and respiratory symptoms. The partial narrowing or occlusion of the upper airway during sleep, followed by an increase in intrathoracic pressure, might predispose the patient to nocturnal GORD and, consequently, to respiratory symptoms.
• Micro-aspiration of stomach acid, or reflux of stomach acid into an inflamed lower oesophagus, might lead to bronchospasm in some patients with asthma. GORD is a common cause of cough and may be associated with poor asthma control.

**Treatment considerations in patients with GORD and asthma**

- A trial of acid suppression therapy may be worthwhile if GORD is suspected, but treatment for reflux does not predictably improve asthma control.29
- GORD may be exacerbated by high doses of beta-agonists or theophylline.

**Obesity**

- Obesity has been identified as a risk factor for asthma in both children and adults.20 However, the findings of a recent study suggest that obesity is positively associated with a history of ever experiencing wheeze in the past, but not with the medical diagnosis of asthma or bronchial hyperresponsiveness.30 Accordingly, wheeze associated with obesity might be due to mechanical factors affecting lung or upper airways, and not with asthma.
- Obesity is common in patients with OSA or GORD.
- In obese patients, OSA may resolve after significant weight loss.

**Asthma and mental illness**

**Practice points**

- When caring for patients with asthma, be alert to the increased risk of comorbid mental illness. (III-3)
- When managing asthma in children, also consider carers’ mental health status, since carers with depression and anxiety use emergency services more often. (III-3)

There has been a recent resurgence of research interest in associations between asthma and mental health. However, there remains an urgent need for further research in some important areas:

- The influence of depression on asthma, and particularly the effect of treating depression in patients with asthma.
- The influence of anxiety on the diagnosis, progression and management of asthma.

**Asthma is not a psychosomatic illness**

The older view that asthma represented a psychosomatic illness and could be treated by psychoanalysis,31 is not supported by current evidence.

**Practice tip**

Consider the potential existence and effects of comorbid depression or anxiety in patients with asthma, especially adolescents.

**Asthma and depression**

- Prevalence estimates for depression among patients with asthma vary between studies. Recent community-based cross-sectional studies suggest that the prevalence of depression is increased among adults with asthma, compared with the general adult population.32–34
- In adolescents with asthma, increased rates of anxiety, but not depression, have been observed.35
- Some studies have reported a correlation between asthma severity and depression rates,36 but others have not observed this association.37,38 Patients’ perceived level of asthma severity may be more closely associated with depression,39,40 but this has not been consistently demonstrated.
- Dyspnoea, wakening at night with asthma symptoms, and morning symptoms were identified as the symptoms most strongly associated with depression in an Australian population.33
- Long-term systemic corticosteroid therapy has been associated with a reduction in serotonin levels, and an increased risk of depression.41,42 However, short courses of oral corticosteroids in asthma might not cause depression or worsen existing depression.43

**Does treatment for depression affect asthma outcomes?**

- There is insufficient evidence from clinical trials to show whether or not treatment for depression affects asthma outcomes.
- Studies of psychotherapy in adults have reported inconsistent results, and there is insufficient evidence for recommendations on the value of psychotherapy in patients with asthma.44
- The combination of asthma and psychological distress clearly has a significant detrimental effect on quality of life.45 Accordingly, recognition and treatment of depression in patients with asthma would be expected to improve quality of life significantly.
Other comorbidities

Asthma and anxiety disorders

- An increased prevalence of anxiety disorders has been demonstrated among patients with asthma.\textsuperscript{32,46} The most common disorders seen are panic attacks, panic disorder and (less commonly) specific phobias and social anxiety disorder.
- People who experience panic attacks are likely to develop asthma, and people with asthma are likely to develop panic attacks.\textsuperscript{47}
- Patients with asthma and anxiety feel less confident in their management and are more likely to seek emergency care.\textsuperscript{48}
- The prevalence of dysfunctional breathing (hyperventilation syndrome or behavioural breathlessness) appears to be high among patients diagnosed with asthma.\textsuperscript{49} This suggests that anxiety-related breathing difficulties may influence the diagnosis of asthma.

Mental illness and adherence to treatment

As would be expected, the presence of depression has a significant impact on adherence to asthma treatment.
- Investigators in one study using computerised inhalation devices found that more than half of participating patients took less than 70% of their inhaled medication. Mean depression scores were higher among patients who showed low adherence than among adherent patients.\textsuperscript{50} Whether depression results in poor adherence, or poor control results in depression, has not been established.
- Anxiety has not been associated with poor treatment adherence and, theoretically, anxiety might be expected to promote adherence. Anxiety may promote help-seeking behaviour in patients when self-management would be preferable. However, anxiety might also improve self care and therefore reduce the need for medical help after an acute attack.\textsuperscript{48}

Mental illness and smoking

- An association between depression and smoking has been established in cross-sectional studies.\textsuperscript{51} The prevalence of depression may be higher in smokers with chronic lung diseases than in smokers with other chronic diseases or no chronic disease.\textsuperscript{34}
- There is no evidence that nicotine replacement therapy is any less effective in depressed smokers than those without depression.\textsuperscript{52}

Mental illness and children with asthma

Anxiety disorders in children with asthma

Asthma in children is strongly associated with anxiety disorders.\textsuperscript{35,46,53} The prevalence of anxiety disorders is higher among children with asthma than among those other chronic diseases.\textsuperscript{54,55}
- The anxiety disorders most commonly associated with asthma are panic disorder and, to a lesser extent, other panic attacks.\textsuperscript{47,56} Patients with asthma are likely to develop panic disorder and patients with panic disorder are likely to develop asthma.\textsuperscript{47}
- The use of high-dose oral corticosteroids has been associated with an increased prevalence of anxiety and aggressive behaviour,\textsuperscript{57} but this association has not been reported for inhaled corticosteroids.

Caregivers’ mental health

In dealing with children, recognise the potential impact of family issues including caregiver affect, on the child’s health.
- Studies investigating the impact of caregiver depression on children’s asthma have reported inconsistent findings. Poorer asthma outcomes have been reported among children whose mothers are affected by depression or life stressors,\textsuperscript{58} whereas other investigators have found mothers’ depression affects treatment adherence and illness management but not asthma morbidity.\textsuperscript{59}
- Caregiver affect has been shown to strongly influence health care utilisation for children’s asthma, as well as having an impact on the caregivers’ quality of life.\textsuperscript{60} The provision of a support program for the families of children moderate-to-severe asthma has been shown to reduce mothers’ anxiety levels.\textsuperscript{61}
- Family therapy for families of children with asthma may improve the child’s asthma.\textsuperscript{62}

Key points

- There are strong associations between asthma and mental illness, but it is not clear whether treatment for mental illness affects symptoms or control of asthma.
- Patients with depression and asthma have poorer quality of life than patients with either alone.
- The mental health of parents and carers of children with asthma significantly affects utilisation of services.
- Patients with chronic lung disease who also have depression are more likely to smoke than patients with chronic lung disease only.
## Prevention of asthma

<table>
<thead>
<tr>
<th>SUMMARY OF PRACTICE POINTS</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>A smoke-free environment should be recommended for all children, and all pregnant and breastfeeding women should be advised not to smoke.</td>
<td>✓</td>
</tr>
<tr>
<td>Avoidance of environmental tobacco smoke may reduce the risk of childhood asthma.</td>
<td>III-2</td>
</tr>
<tr>
<td>Breastfeeding may lower asthma risk during early childhood, but does not provide long-term asthma protection.</td>
<td>III-2</td>
</tr>
<tr>
<td>If breastfeeding is not possible, infant feeding with hydrolysed milk formulae may slightly lower the risk of childhood allergy, wheezing and asthma, compared with use of other formulae.</td>
<td>I</td>
</tr>
<tr>
<td>Infant feeding with soy formulae does not prevent asthma.</td>
<td>I</td>
</tr>
<tr>
<td>Omega-3 fatty acid supplementation in at-risk infants does not appear to reduce the risk of childhood asthma or wheezing.</td>
<td>II</td>
</tr>
<tr>
<td>Probiotic supplementation of mothers during late pregnancy and lactation, or of non-breastfed infants, does not appear to prevent asthma.</td>
<td>II</td>
</tr>
<tr>
<td>Avoidance of commonly allergenic foods during pregnancy or lactation has no effect on the development of childhood asthma.</td>
<td>I</td>
</tr>
<tr>
<td>Avoidance of commonly allergenic foods in infant diets does not reduce the risk of childhood asthma.</td>
<td>II</td>
</tr>
<tr>
<td>Measures to reduce exposure to dust mite do not appear to decrease the rates of asthma or wheeze in young children.</td>
<td>II</td>
</tr>
<tr>
<td>On current evidence, advising families to avoid exposure to pets is not warranted.</td>
<td>✓</td>
</tr>
<tr>
<td>Multifaceted environmental controls that include allergen avoidance, undertaken during infancy, may reduce asthma symptoms in young children but have no effect on lung function or bronchial hyperresponsiveness.</td>
<td>II</td>
</tr>
<tr>
<td>Long-term treatment with antihistamines does not reduce the risk of asthma developing in children with atopic dermatitis, including those who are sensitive to house dust mite and/or grass pollen.</td>
<td>II</td>
</tr>
<tr>
<td>Immunotherapy may reduce asthma risk in children with seasonal allergic rhinoconjunctivitis.</td>
<td>II</td>
</tr>
</tbody>
</table>
People consulting health professionals often ask about their risk or their children’s risk of developing asthma, how their family history affects asthma risk, and whether they can do anything to prevent their child developing asthma. The development of asthma involves a complex interaction of genetic factors and environmental influences. A family history of allergy and asthma can be used to identify children at increased risk of asthma.

There has been considerable interest in whether a child’s likelihood of developing asthma may be reduced by changing the environment. Much of the evidence for benefits of proposed environmental modifications comes from epidemiological studies. Interventional studies have examined the effect of manipulating various environmental factors, but as yet there is little convincing evidence that specific interventions are highly effective in preventing the onset of asthma.

Primary prevention: can the onset of asthma be prevented?

Advice for parents and carers is summarised in Table 1.

Does exposure to environmental tobacco smoke increase asthma risk?

A smoke-free environment should be recommended for all children, and all pregnant women should be advised not to smoke.

• The link between exposure to environmental tobacco smoke (ETS) during foetal development and early childhood, and increased risk of respiratory illnesses including asthma, has been well documented in epidemiological studies.¹
  • Prenatal exposure may carry the greater risk.¹
  • Genetic factors appear to influence the effects of ETS on the risk of developing asthma.²
  • Exposure to ETS appears to be an independent risk factor for allergic sensitisation also.³

In the absence of randomised controlled trials assessing the effect of ETS avoidance on the risk of developing asthma, avoidance of ETS should be recommended, based on epidemiological evidence that it increases asthma risk, as well as other known detrimental effects. For information on smoking-related risks and smoking cessation, see Smoking and asthma.

Does infant feeding affect asthma risk?

Breastfeeding

While breastfeeding should be promoted for its many beneficial effects, the evidence that it may prevent the development of asthma is conflicting.

• Clinical studies have demonstrated that breastfeeding confers a small protective effect against asthma risk, especially in children with a family history of atopy.⁴
• A large Australian prospective cohort study in children aged 6 years found that the introduction of milk other than breast milk before age 4 months was a significant risk factor for asthma (odds ratio 1.25; CI 1.02–1.52), wheezing and atopy.⁵
• Breastfeeding appears to delay the onset of asthma and recurrent wheeze or actively protect children less than 24 months old against asthma and recurrent wheeze, and might reduce the prevalence of asthma and recurrent wheeze in children exposed to environmental tobacco smoke. However, some studies suggest that the protective effect of breastfeeding against wheezing is strongest in non-atopic children, and may be mainly due to prevention of wheezing during viral respiratory infections, rather than an effect on the development of asthma.\textsuperscript{7,8}

• There is no good evidence that the protective influence of breastfeeding seen in some studies in early childhood extends into later childhood or adult life. Some studies have suggested that breastfeeding, particularly in atopic children, may be associated with an increased risk of asthma development in later childhood and adulthood.\textsuperscript{8,9}

Infant feed formulae

• Hydrolysed milk formulae: there is some evidence that infant feeding with hydrolysed milk formulae is protective against allergy, wheezing and asthma,\textsuperscript{10,11} but less protective than breastfeeding. The evidence is stronger for an effect on infant wheezing\textsuperscript{10} than on asthma.

• Soy formulae: infant feeding with soy formulae does not appear to prevent allergy or asthma.\textsuperscript{12}

Fish oil supplementation

• Epidemiological evidence suggests that a diet rich in oily fish is associated with a decreased likelihood of developing asthma.

• However, a randomised controlled clinical trial observed no reduction in prevalences of childhood asthma or wheezing following supplementation with omega-3 fatty acids in at-risk infants. Omega-3 fatty acid supplementation was associated with a reduction in cough in atopic children at age 3 years and at age 5 years.\textsuperscript{13,14}

Probiotics

A randomised controlled clinical trial of probiotic supplementation in at-risk families (mothers during late pregnancy and lactation and non-breastfed infants during the first 6 months of life) demonstrated a reduction in eczema at 2 and 4 years, but no effect on allergen sensitisation or asthma.\textsuperscript{15,16}

Does allergen avoidance reduce asthma risk?

**Practice points**

- Avoidance of commonly allergenic foods during pregnancy or lactation has no effect on the development of childhood asthma. (I)
- Avoidance of commonly allergenic foods in infant diets does not reduce the risk of childhood asthma. (II)
- Measures to reduce exposure to dust mite do not appear to decrease the rates of asthma or wheeze in young children. (II)
- On current evidence, advising families to avoid exposure to pets is not warranted. ✓
- Multifaceted environmental controls that include allergen avoidance, undertaken during infancy, may reduce asthma symptoms in young children but have no effect on lung function or bronchial hyperresponsiveness. (II)

Maternal dietary allergen avoidance in pregnancy or lactation

Maternal dietary allergen avoidance during pregnancy or lactation appears to have no effect on the child’s risk of developing asthma.\textsuperscript{17}

Dietary allergen avoidance in infancy

Combined maternal and infant food allergen avoidance in at-risk families does not appear to reduce rates of asthma or allergic rhinitis by age 7 years.\textsuperscript{18}

Dust mite avoidance

To date, randomised controlled clinical trials assessing the effect of house dust mite avoidance in at-risk families have not demonstrated substantial reductions in asthma.

• An Australian study of dust mite avoidance has not demonstrated any significant reduction in asthma, wheeze or cough by 3 years of age or by 5 years of age, despite a modest reduction in sensitisation to house dust mite.\textsuperscript{13,14}

• A UK study of dust mite avoidance has demonstrated significantly better lung function at age 3 years but no significant difference in asthma or wheeze, and an increased risk of mite sensitisation.\textsuperscript{19} Two other European studies have not shown any reduction in mite sensitisation or the development of asthma in the first 2 years of life.\textsuperscript{20,21}
Pet allergen avoidance

Firm recommendations about exposure to pets cannot be made because of conflicting epidemiological data on the effect of early pet avoidance on asthma development.

- A systematic review concluded that exposure to pets increases the risk of asthma in children older than 6 years, but other studies have suggested that early pet exposure may protect against asthma development.
- The issue is complex; any protective effects associated with animal exposure prior to the development of asthma may be complicated by sensitisation and triggering of symptoms in those who go on to develop asthma.

Multifaceted interventions including allergen avoidance

Rates of childhood asthma might be reduced by families avoiding exposure to multiple risk factors, including allergens.

- A UK randomised controlled trial of a combined dietary allergen and house dust mite avoidance intervention in at-risk infants in the first 9 months of life has demonstrated a significant reduction in asthma symptoms and atopic sensitisation at age 8 years, but no significant effect on lung function or the prevalence of bronchial hyperresponsiveness.
- A Canadian randomised controlled trial of a multifaceted intervention in at-risk infants in the first year of life (avoidance of house dust mite, pets, environmental tobacco smoke, encouragement of breastfeeding and delayed introduction of solid feeds) demonstrated a reduction in asthma symptoms but no effect on bronchial hyperresponsiveness.

For more information on strategies to reduce allergen exposure, see Asthma and Allergy.

Other environmental factors

The observation among some communities that children living on farms show relatively low levels of allergic disease including asthma has led to the hypothesis that high exposure to bacterial endotoxins and lipopolysaccharides may reduce asthma risk.

Table 1. What can health professionals advise families about preventing asthma in infants and unborn babies?

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Advice based on current evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergen avoidance in pregnancy</td>
<td>• Dietary restrictions in pregnancy are not recommended.</td>
</tr>
<tr>
<td></td>
<td>• Aeroallergen avoidance in pregnancy has not been shown to reduce allergic disease, and is not recommended.</td>
</tr>
<tr>
<td>Breastfeeding</td>
<td>• Breastfeeding should be recommended.</td>
</tr>
<tr>
<td></td>
<td>• Maternal dietary restrictions during breastfeeding are not recommended.</td>
</tr>
<tr>
<td>Infant formulae</td>
<td>• The use of formulae is recommended only in infants who have a high risk for asthma and if breastfeeding is not possible.</td>
</tr>
<tr>
<td></td>
<td>• A partially or extensively hydrolysed formula may reduce the risk of developing asthma.</td>
</tr>
<tr>
<td></td>
<td>• Soy formulae and other formulae (e.g. goat's milk) are not recommended for the reduction of asthma risk.</td>
</tr>
<tr>
<td>Infant diet</td>
<td>• There is no evidence that avoidance of commonly allergenic foods prevents the development of asthma.</td>
</tr>
<tr>
<td></td>
<td>• There is no evidence at present to suggest that supplementation with fish oil or probiotics prevents the development of asthma.</td>
</tr>
<tr>
<td>Avoidance of house dust mite exposure</td>
<td>• There is no evidence to suggest that house dust mite reduction alone prevents the development of asthma in Australia.</td>
</tr>
<tr>
<td></td>
<td>• Multifaceted interventions that include house dust mite avoidance have been shown to reduce asthma symptoms, but have not influenced lung function or prevalence of bronchial hyperresponsiveness.</td>
</tr>
<tr>
<td>Pet exposure</td>
<td>On current evidence, advising families to avoid exposure to pets is not warranted.</td>
</tr>
<tr>
<td>Smoking and other irritants</td>
<td>Pregnant women should be advised not to smoke during pregnancy. Parents should be advised not to smoke, and to avoid children being exposed to other people’s smoke.</td>
</tr>
</tbody>
</table>

Adapted from Prescott SL, Tang M, 2004
Secondary prevention: can asthma be prevented in patients with other atopic disease?

**Practice points**
- Long-term treatment with antihistamines does not reduce the risk of asthma developing in children with atopic dermatitis, including those who are sensitive to house dust mite and/or grass pollen. (II)
- Immunotherapy may reduce asthma risk in children with seasonal allergic rhinoconjunctivitis. (II)

**Children**

**Avoidance of allergens and environmental tobacco smoke**
No studies have assessed whether the avoidance of allergens or exposure to ETS is effective in reducing the risk of asthma developing in children with other manifestations of atopy.

Because of other known adverse effects of ETS, a smoke-free environment should be recommended for all children.

**Immunotherapy**
In children with seasonal allergic rhinoconjunctivitis, immunotherapy for house dust mite or pollen allergies may reduce their risk of developing asthma. See Asthma and allergy.

**Drug therapy**
- A 1-year randomised controlled clinical trial of ketotifen in children with atopic dermatitis but no history of asthma demonstrated a significant reduction in asthma symptoms during the year of therapy, but it is difficult to know whether this was a true preventive effect or reflected control of wheezing symptoms.
- Despite earlier findings that suggested cetirizine treatment may reduce risk of developing asthma among infants with atopic dermatitis and sensitivity to house dust mite or grass pollen, a recent randomised controlled clinical trial of cetirizine observed no reduction in asthma onset in this subgroup.

**Adults**

**Smoking**
Cigarette smoking increases the risk of developing asthma in response to frequent exposure to some sensitising agents associated with occupational asthma.

**Occupational asthma**
- Reducing airborne exposure to potential allergens in the workplace lowers workers’ risk for becoming sensitised and developing occupational asthma.
- The correct use of respiratory protective equipment reduces the risk of occupational asthma but does not completely prevent it.

See Occupational asthma.

**Tertiary prevention: can asthma be ‘cured’?**
Some forms of childhood asthma (e.g. wheezing associated with upper respiratory tract viral infections) may resolve spontaneously, and established asthma may show periods of apparent symptomatic remission. However, there is currently no evidence to suggest that established atopic asthma can be reversed.

Patients and carers should be assured that asthma can usually be effectively controlled by appropriate management.
Respiratory function tables

Mean predicted normal values for healthy adults

The mean predicted normal values (FEV₁, FVC, FEV₁/FVC) for adult Caucasian males (aged 20-80 years) and females (aged 18-80 years) are given in the following tables. Contact your local lung function laboratory for advice about predicted values, including lower limits of normal and the effect of ethnicity.


### FEV₁ (L) Male

<table>
<thead>
<tr>
<th>Age</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>145 cm</td>
<td>3.19</td>
<td>3.08</td>
<td>2.97</td>
<td>2.85</td>
<td>2.72</td>
<td>2.58</td>
<td>2.44</td>
<td>2.28</td>
<td>2.12</td>
<td>1.94</td>
<td>1.76</td>
<td>1.57</td>
<td>1.37</td>
</tr>
<tr>
<td>150 cm</td>
<td>3.40</td>
<td>3.29</td>
<td>3.18</td>
<td>3.06</td>
<td>2.93</td>
<td>2.79</td>
<td>2.64</td>
<td>2.49</td>
<td>2.32</td>
<td>2.15</td>
<td>1.97</td>
<td>1.78</td>
<td>1.58</td>
</tr>
<tr>
<td>155 cm</td>
<td>3.61</td>
<td>3.51</td>
<td>3.39</td>
<td>3.27</td>
<td>3.14</td>
<td>3.01</td>
<td>2.86</td>
<td>2.70</td>
<td>2.54</td>
<td>2.37</td>
<td>2.19</td>
<td>2.00</td>
<td>1.80</td>
</tr>
<tr>
<td>160 cm</td>
<td>3.83</td>
<td>3.73</td>
<td>3.62</td>
<td>3.50</td>
<td>3.37</td>
<td>3.23</td>
<td>3.08</td>
<td>2.93</td>
<td>2.76</td>
<td>2.59</td>
<td>2.41</td>
<td>2.22</td>
<td>2.02</td>
</tr>
<tr>
<td>165 cm</td>
<td>4.06</td>
<td>3.96</td>
<td>3.85</td>
<td>3.73</td>
<td>3.60</td>
<td>3.46</td>
<td>3.31</td>
<td>3.15</td>
<td>2.99</td>
<td>2.82</td>
<td>2.65</td>
<td>2.45</td>
<td>2.25</td>
</tr>
<tr>
<td>170 cm</td>
<td>4.30</td>
<td>4.19</td>
<td>4.08</td>
<td>3.96</td>
<td>3.83</td>
<td>3.69</td>
<td>3.55</td>
<td>3.39</td>
<td>3.23</td>
<td>3.05</td>
<td>2.87</td>
<td>2.68</td>
<td>2.48</td>
</tr>
<tr>
<td>175 cm</td>
<td>4.54</td>
<td>4.44</td>
<td>4.33</td>
<td>4.20</td>
<td>4.07</td>
<td>3.94</td>
<td>3.79</td>
<td>3.63</td>
<td>3.47</td>
<td>3.30</td>
<td>3.12</td>
<td>2.93</td>
<td>2.73</td>
</tr>
<tr>
<td>180 cm</td>
<td>4.79</td>
<td>4.69</td>
<td>4.58</td>
<td>4.45</td>
<td>4.32</td>
<td>4.19</td>
<td>4.04</td>
<td>3.88</td>
<td>3.72</td>
<td>3.55</td>
<td>3.37</td>
<td>3.18</td>
<td>2.98</td>
</tr>
<tr>
<td>185 cm</td>
<td>5.05</td>
<td>4.95</td>
<td>4.83</td>
<td>4.71</td>
<td>4.58</td>
<td>4.44</td>
<td>4.30</td>
<td>4.14</td>
<td>3.98</td>
<td>3.80</td>
<td>3.62</td>
<td>3.43</td>
<td>3.24</td>
</tr>
<tr>
<td>190 cm</td>
<td>5.31</td>
<td>5.21</td>
<td>5.10</td>
<td>4.98</td>
<td>4.85</td>
<td>4.71</td>
<td>4.56</td>
<td>4.41</td>
<td>4.24</td>
<td>4.07</td>
<td>3.89</td>
<td>3.70</td>
<td>3.50</td>
</tr>
<tr>
<td>195 cm</td>
<td>5.58</td>
<td>5.48</td>
<td>5.37</td>
<td>5.25</td>
<td>5.12</td>
<td>4.98</td>
<td>4.83</td>
<td>4.68</td>
<td>4.51</td>
<td>4.34</td>
<td>4.16</td>
<td>3.97</td>
<td>3.77</td>
</tr>
</tbody>
</table>

### FVC (L) Male

<table>
<thead>
<tr>
<th>Age</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>145 cm</td>
<td>3.63</td>
<td>3.57</td>
<td>3.50</td>
<td>3.42</td>
<td>3.32</td>
<td>3.21</td>
<td>3.09</td>
<td>2.95</td>
<td>2.80</td>
<td>2.63</td>
<td>2.45</td>
<td>2.26</td>
<td>2.06</td>
</tr>
<tr>
<td>150 cm</td>
<td>3.91</td>
<td>3.85</td>
<td>3.78</td>
<td>3.69</td>
<td>3.60</td>
<td>3.49</td>
<td>3.36</td>
<td>3.22</td>
<td>3.07</td>
<td>2.91</td>
<td>2.73</td>
<td>2.54</td>
<td>2.33</td>
</tr>
<tr>
<td>155 cm</td>
<td>4.19</td>
<td>4.13</td>
<td>4.06</td>
<td>3.98</td>
<td>3.88</td>
<td>3.77</td>
<td>3.64</td>
<td>3.51</td>
<td>3.36</td>
<td>3.19</td>
<td>3.01</td>
<td>2.82</td>
<td>2.62</td>
</tr>
<tr>
<td>160 cm</td>
<td>4.48</td>
<td>4.43</td>
<td>4.36</td>
<td>4.27</td>
<td>4.17</td>
<td>4.06</td>
<td>3.94</td>
<td>3.80</td>
<td>3.65</td>
<td>3.48</td>
<td>3.31</td>
<td>3.11</td>
<td>2.91</td>
</tr>
<tr>
<td>165 cm</td>
<td>4.79</td>
<td>4.73</td>
<td>4.66</td>
<td>4.57</td>
<td>4.48</td>
<td>4.37</td>
<td>4.24</td>
<td>4.10</td>
<td>3.95</td>
<td>3.79</td>
<td>3.61</td>
<td>3.42</td>
<td>3.21</td>
</tr>
<tr>
<td>170 cm</td>
<td>5.10</td>
<td>5.04</td>
<td>4.97</td>
<td>4.89</td>
<td>4.79</td>
<td>4.68</td>
<td>4.55</td>
<td>4.42</td>
<td>4.26</td>
<td>4.10</td>
<td>3.92</td>
<td>3.73</td>
<td>3.52</td>
</tr>
<tr>
<td>175 cm</td>
<td>5.42</td>
<td>5.36</td>
<td>5.29</td>
<td>5.21</td>
<td>5.11</td>
<td>5.00</td>
<td>4.88</td>
<td>4.74</td>
<td>4.59</td>
<td>4.42</td>
<td>4.24</td>
<td>4.05</td>
<td>3.85</td>
</tr>
<tr>
<td>180 cm</td>
<td>5.75</td>
<td>5.69</td>
<td>5.62</td>
<td>5.54</td>
<td>5.44</td>
<td>5.33</td>
<td>5.21</td>
<td>5.07</td>
<td>4.92</td>
<td>4.75</td>
<td>4.57</td>
<td>4.38</td>
<td>4.18</td>
</tr>
<tr>
<td>185 cm</td>
<td>6.09</td>
<td>6.03</td>
<td>5.96</td>
<td>5.88</td>
<td>5.78</td>
<td>5.67</td>
<td>5.55</td>
<td>5.41</td>
<td>5.26</td>
<td>5.09</td>
<td>4.91</td>
<td>4.72</td>
<td>4.52</td>
</tr>
<tr>
<td>190 cm</td>
<td>6.44</td>
<td>6.38</td>
<td>6.31</td>
<td>6.23</td>
<td>6.13</td>
<td>6.02</td>
<td>5.90</td>
<td>5.76</td>
<td>5.61</td>
<td>5.44</td>
<td>5.26</td>
<td>5.07</td>
<td>4.87</td>
</tr>
<tr>
<td>195 cm</td>
<td>6.80</td>
<td>6.74</td>
<td>6.67</td>
<td>6.59</td>
<td>6.49</td>
<td>6.38</td>
<td>6.25</td>
<td>6.12</td>
<td>5.97</td>
<td>5.80</td>
<td>5.62</td>
<td>5.43</td>
<td>5.22</td>
</tr>
</tbody>
</table>

### FEV₁/FVC (%) Male

<table>
<thead>
<tr>
<th>Age</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Heights</td>
<td>83.9</td>
<td>82.9</td>
<td>81.9</td>
<td>80.8</td>
<td>79.8</td>
<td>78.8</td>
<td>77.7</td>
<td>76.7</td>
<td>75.7</td>
<td>74.6</td>
<td>73.6</td>
<td>72.6</td>
<td>71.5</td>
</tr>
</tbody>
</table>
### FEV1 (L) Female

<table>
<thead>
<tr>
<th>Age</th>
<th>18</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>145 cm</td>
<td>2.72</td>
<td>2.70</td>
<td>2.64</td>
<td>2.57</td>
<td>2.49</td>
<td>2.40</td>
<td>2.30</td>
<td>2.18</td>
<td>2.06</td>
<td>1.94</td>
<td>1.80</td>
<td>1.65</td>
<td>1.49</td>
<td>1.32</td>
</tr>
<tr>
<td>150 cm</td>
<td>2.89</td>
<td>2.87</td>
<td>2.81</td>
<td>2.74</td>
<td>2.66</td>
<td>2.57</td>
<td>2.46</td>
<td>2.35</td>
<td>2.23</td>
<td>2.10</td>
<td>1.97</td>
<td>1.82</td>
<td>1.66</td>
<td>1.49</td>
</tr>
<tr>
<td>155 cm</td>
<td>3.07</td>
<td>3.05</td>
<td>2.98</td>
<td>2.91</td>
<td>2.83</td>
<td>2.74</td>
<td>2.64</td>
<td>2.53</td>
<td>2.41</td>
<td>2.28</td>
<td>2.14</td>
<td>1.99</td>
<td>1.83</td>
<td>1.66</td>
</tr>
<tr>
<td>160 cm</td>
<td>3.25</td>
<td>3.23</td>
<td>3.16</td>
<td>3.09</td>
<td>3.01</td>
<td>2.92</td>
<td>2.82</td>
<td>2.71</td>
<td>2.59</td>
<td>2.46</td>
<td>2.32</td>
<td>2.17</td>
<td>2.01</td>
<td>1.85</td>
</tr>
<tr>
<td>165 cm</td>
<td>3.44</td>
<td>3.41</td>
<td>3.35</td>
<td>3.28</td>
<td>3.20</td>
<td>3.11</td>
<td>3.01</td>
<td>2.90</td>
<td>2.78</td>
<td>2.65</td>
<td>2.51</td>
<td>2.36</td>
<td>2.20</td>
<td>2.03</td>
</tr>
<tr>
<td>170 cm</td>
<td>3.63</td>
<td>3.61</td>
<td>3.54</td>
<td>3.47</td>
<td>3.39</td>
<td>3.30</td>
<td>3.20</td>
<td>3.09</td>
<td>2.97</td>
<td>2.84</td>
<td>2.70</td>
<td>2.55</td>
<td>2.39</td>
<td>2.23</td>
</tr>
<tr>
<td>175 cm</td>
<td>3.83</td>
<td>3.80</td>
<td>3.74</td>
<td>3.67</td>
<td>3.59</td>
<td>3.50</td>
<td>3.40</td>
<td>3.29</td>
<td>3.17</td>
<td>3.04</td>
<td>2.90</td>
<td>2.75</td>
<td>2.59</td>
<td>2.42</td>
</tr>
<tr>
<td>180 cm</td>
<td>4.03</td>
<td>4.01</td>
<td>3.95</td>
<td>3.88</td>
<td>3.79</td>
<td>3.70</td>
<td>3.60</td>
<td>3.49</td>
<td>3.37</td>
<td>3.24</td>
<td>3.10</td>
<td>2.95</td>
<td>2.80</td>
<td>2.63</td>
</tr>
<tr>
<td>185 cm</td>
<td>4.24</td>
<td>4.22</td>
<td>4.16</td>
<td>4.08</td>
<td>4.00</td>
<td>3.91</td>
<td>3.81</td>
<td>3.70</td>
<td>3.58</td>
<td>3.45</td>
<td>3.31</td>
<td>3.16</td>
<td>3.01</td>
<td>2.84</td>
</tr>
<tr>
<td>190 cm</td>
<td>4.46</td>
<td>4.43</td>
<td>4.37</td>
<td>4.30</td>
<td>4.22</td>
<td>4.13</td>
<td>4.03</td>
<td>3.92</td>
<td>3.80</td>
<td>3.67</td>
<td>3.53</td>
<td>3.38</td>
<td>3.22</td>
<td>3.05</td>
</tr>
<tr>
<td>195 cm</td>
<td>4.68</td>
<td>4.65</td>
<td>4.59</td>
<td>4.52</td>
<td>4.44</td>
<td>4.35</td>
<td>4.25</td>
<td>4.14</td>
<td>4.02</td>
<td>3.89</td>
<td>3.75</td>
<td>3.60</td>
<td>3.44</td>
<td>3.27</td>
</tr>
</tbody>
</table>

### FVC (L) Female

<table>
<thead>
<tr>
<th>Age</th>
<th>18</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>145 cm</td>
<td>2.97</td>
<td>2.98</td>
<td>2.99</td>
<td>2.98</td>
<td>2.95</td>
<td>2.90</td>
<td>2.83</td>
<td>2.74</td>
<td>2.63</td>
<td>2.51</td>
<td>2.36</td>
<td>2.20</td>
<td>2.01</td>
<td>1.81</td>
</tr>
<tr>
<td>150 cm</td>
<td>3.19</td>
<td>3.20</td>
<td>3.21</td>
<td>3.19</td>
<td>3.16</td>
<td>3.11</td>
<td>3.05</td>
<td>2.96</td>
<td>2.85</td>
<td>2.72</td>
<td>2.58</td>
<td>2.41</td>
<td>2.23</td>
<td>2.03</td>
</tr>
<tr>
<td>155 cm</td>
<td>3.42</td>
<td>3.42</td>
<td>3.43</td>
<td>3.42</td>
<td>3.39</td>
<td>3.34</td>
<td>3.27</td>
<td>3.18</td>
<td>3.08</td>
<td>2.95</td>
<td>2.80</td>
<td>2.64</td>
<td>2.46</td>
<td>2.25</td>
</tr>
<tr>
<td>160 cm</td>
<td>3.65</td>
<td>3.66</td>
<td>3.67</td>
<td>3.65</td>
<td>3.62</td>
<td>3.57</td>
<td>3.50</td>
<td>3.42</td>
<td>3.31</td>
<td>3.18</td>
<td>3.04</td>
<td>2.87</td>
<td>2.69</td>
<td>2.49</td>
</tr>
<tr>
<td>165 cm</td>
<td>3.89</td>
<td>3.90</td>
<td>3.91</td>
<td>3.89</td>
<td>3.86</td>
<td>3.81</td>
<td>3.75</td>
<td>3.66</td>
<td>3.55</td>
<td>3.42</td>
<td>3.28</td>
<td>3.11</td>
<td>2.93</td>
<td>2.73</td>
</tr>
<tr>
<td>180 cm</td>
<td>4.66</td>
<td>4.67</td>
<td>4.67</td>
<td>4.66</td>
<td>4.63</td>
<td>4.58</td>
<td>4.51</td>
<td>4.42</td>
<td>4.32</td>
<td>4.19</td>
<td>4.05</td>
<td>3.88</td>
<td>3.70</td>
<td>3.50</td>
</tr>
<tr>
<td>185 cm</td>
<td>4.93</td>
<td>4.94</td>
<td>4.94</td>
<td>4.93</td>
<td>4.90</td>
<td>4.85</td>
<td>4.78</td>
<td>4.69</td>
<td>4.59</td>
<td>4.46</td>
<td>4.32</td>
<td>4.15</td>
<td>3.97</td>
<td>3.77</td>
</tr>
<tr>
<td>190 cm</td>
<td>5.21</td>
<td>5.21</td>
<td>5.22</td>
<td>5.21</td>
<td>5.18</td>
<td>5.13</td>
<td>5.06</td>
<td>4.97</td>
<td>4.87</td>
<td>4.74</td>
<td>4.59</td>
<td>4.43</td>
<td>4.25</td>
<td>4.04</td>
</tr>
<tr>
<td>195 cm</td>
<td>5.49</td>
<td>5.50</td>
<td>5.51</td>
<td>5.49</td>
<td>5.46</td>
<td>5.41</td>
<td>5.35</td>
<td>5.26</td>
<td>5.15</td>
<td>5.02</td>
<td>4.88</td>
<td>4.71</td>
<td>4.53</td>
<td>4.33</td>
</tr>
</tbody>
</table>

### FEV1/FVC (%) Female

<table>
<thead>
<tr>
<th>Age</th>
<th>18</th>
<th>20</th>
<th>25</th>
<th>30</th>
<th>35</th>
<th>40</th>
<th>45</th>
<th>50</th>
<th>55</th>
<th>60</th>
<th>65</th>
<th>70</th>
<th>75</th>
<th>80</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Heights</td>
<td>87.0</td>
<td>86.6</td>
<td>85.5</td>
<td>84.4</td>
<td>83.4</td>
<td>82.3</td>
<td>81.2</td>
<td>80.2</td>
<td>79.1</td>
<td>78.1</td>
<td>77.0</td>
<td>75.9</td>
<td>74.9</td>
<td>73.8</td>
</tr>
</tbody>
</table>
Respiratory function tables

Mean predicted normal values for healthy children and adolescents

The mean predicted normal values (FEV₁, FVC, FEV₁/FVC, PEF) for Caucasian children and adolescents (males aged 8-20 years and females aged 8-18 years) are given in the following tables. Contact your local lung function laboratory for advice about predicted values, including lower limits of normal and the effect of ethnicity.


FEV₁ (L) Male Children (<20 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>1.42</td>
<td>1.49</td>
<td>1.61</td>
<td>1.76</td>
<td>1.95</td>
<td>2.17</td>
<td>2.43</td>
</tr>
<tr>
<td>130 cm</td>
<td>1.60</td>
<td>1.67</td>
<td>1.79</td>
<td>1.94</td>
<td>2.13</td>
<td>2.35</td>
<td>2.61</td>
</tr>
<tr>
<td>135 cm</td>
<td>1.78</td>
<td>1.86</td>
<td>1.98</td>
<td>2.13</td>
<td>2.31</td>
<td>2.54</td>
<td>2.79</td>
</tr>
<tr>
<td>140 cm</td>
<td>1.98</td>
<td>2.06</td>
<td>2.17</td>
<td>2.32</td>
<td>2.51</td>
<td>2.73</td>
<td>2.99</td>
</tr>
<tr>
<td>145 cm</td>
<td>2.18</td>
<td>2.26</td>
<td>2.37</td>
<td>2.52</td>
<td>2.71</td>
<td>2.93</td>
<td>3.19</td>
</tr>
<tr>
<td>150 cm</td>
<td>2.38</td>
<td>2.46</td>
<td>2.58</td>
<td>2.73</td>
<td>2.92</td>
<td>3.14</td>
<td>3.40</td>
</tr>
<tr>
<td>155 cm</td>
<td>2.60</td>
<td>2.68</td>
<td>2.79</td>
<td>2.94</td>
<td>3.13</td>
<td>3.35</td>
<td>3.61</td>
</tr>
<tr>
<td>160 cm</td>
<td>2.82</td>
<td>2.90</td>
<td>3.02</td>
<td>3.17</td>
<td>3.35</td>
<td>3.58</td>
<td>3.83</td>
</tr>
<tr>
<td>165 cm</td>
<td>3.05</td>
<td>3.13</td>
<td>3.24</td>
<td>3.40</td>
<td>3.58</td>
<td>3.80</td>
<td>4.06</td>
</tr>
<tr>
<td>170 cm</td>
<td>3.29</td>
<td>3.37</td>
<td>3.48</td>
<td>3.63</td>
<td>3.82</td>
<td>4.04</td>
<td>4.30</td>
</tr>
<tr>
<td>175 cm</td>
<td>3.53</td>
<td>3.61</td>
<td>3.72</td>
<td>3.87</td>
<td>4.06</td>
<td>4.28</td>
<td>4.54</td>
</tr>
<tr>
<td>180 cm</td>
<td>3.78</td>
<td>3.86</td>
<td>3.97</td>
<td>4.13</td>
<td>4.31</td>
<td>4.53</td>
<td>4.79</td>
</tr>
<tr>
<td>185 cm</td>
<td>4.04</td>
<td>4.12</td>
<td>4.23</td>
<td>4.38</td>
<td>4.57</td>
<td>4.79</td>
<td>5.05</td>
</tr>
</tbody>
</table>

FVC (L) Male Children (<20 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>1.67</td>
<td>1.63</td>
<td>1.66</td>
<td>1.78</td>
<td>1.98</td>
<td>2.26</td>
<td>2.62</td>
</tr>
<tr>
<td>130 cm</td>
<td>1.91</td>
<td>1.86</td>
<td>1.90</td>
<td>2.02</td>
<td>2.22</td>
<td>2.50</td>
<td>2.86</td>
</tr>
<tr>
<td>135 cm</td>
<td>2.15</td>
<td>2.11</td>
<td>2.15</td>
<td>2.27</td>
<td>2.47</td>
<td>2.75</td>
<td>3.11</td>
</tr>
<tr>
<td>140 cm</td>
<td>2.41</td>
<td>2.37</td>
<td>2.40</td>
<td>2.52</td>
<td>2.72</td>
<td>3.00</td>
<td>3.37</td>
</tr>
<tr>
<td>145 cm</td>
<td>2.68</td>
<td>2.63</td>
<td>2.67</td>
<td>2.79</td>
<td>2.99</td>
<td>3.27</td>
<td>3.63</td>
</tr>
<tr>
<td>150 cm</td>
<td>2.95</td>
<td>2.91</td>
<td>2.95</td>
<td>3.06</td>
<td>3.26</td>
<td>3.54</td>
<td>3.91</td>
</tr>
<tr>
<td>155 cm</td>
<td>3.24</td>
<td>3.19</td>
<td>3.23</td>
<td>3.35</td>
<td>3.55</td>
<td>3.83</td>
<td>4.19</td>
</tr>
<tr>
<td>160 cm</td>
<td>3.53</td>
<td>3.49</td>
<td>3.52</td>
<td>3.64</td>
<td>3.84</td>
<td>4.12</td>
<td>4.48</td>
</tr>
<tr>
<td>165 cm</td>
<td>3.83</td>
<td>3.79</td>
<td>3.83</td>
<td>3.94</td>
<td>4.14</td>
<td>4.43</td>
<td>4.79</td>
</tr>
<tr>
<td>170 cm</td>
<td>4.14</td>
<td>4.10</td>
<td>4.14</td>
<td>4.26</td>
<td>4.46</td>
<td>4.74</td>
<td>5.10</td>
</tr>
<tr>
<td>175 cm</td>
<td>4.47</td>
<td>4.42</td>
<td>4.46</td>
<td>4.58</td>
<td>4.78</td>
<td>5.06</td>
<td>5.42</td>
</tr>
<tr>
<td>180 cm</td>
<td>4.80</td>
<td>4.75</td>
<td>4.79</td>
<td>4.91</td>
<td>5.11</td>
<td>5.39</td>
<td>5.75</td>
</tr>
<tr>
<td>185 cm</td>
<td>5.14</td>
<td>5.09</td>
<td>5.13</td>
<td>5.25</td>
<td>5.45</td>
<td>5.73</td>
<td>6.09</td>
</tr>
</tbody>
</table>
### FEV1/FVC (%) Male Children (<20 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Heights</td>
<td>86.4</td>
<td>86.0</td>
<td>85.6</td>
<td>85.2</td>
<td>84.8</td>
<td>84.3</td>
<td>83.9</td>
</tr>
</tbody>
</table>

### PEF (L/min) Male Children (<20 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
<th>20</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>189</td>
<td>203</td>
<td>223</td>
<td>249</td>
<td>281</td>
<td>320</td>
<td>365</td>
</tr>
<tr>
<td>130 cm</td>
<td>208</td>
<td>222</td>
<td>242</td>
<td>268</td>
<td>300</td>
<td>339</td>
<td>384</td>
</tr>
<tr>
<td>135 cm</td>
<td>228</td>
<td>242</td>
<td>262</td>
<td>288</td>
<td>320</td>
<td>359</td>
<td>404</td>
</tr>
<tr>
<td>140 cm</td>
<td>249</td>
<td>262</td>
<td>282</td>
<td>308</td>
<td>341</td>
<td>380</td>
<td>425</td>
</tr>
<tr>
<td>145 cm</td>
<td>270</td>
<td>284</td>
<td>304</td>
<td>330</td>
<td>362</td>
<td>401</td>
<td>446</td>
</tr>
<tr>
<td>150 cm</td>
<td>292</td>
<td>306</td>
<td>326</td>
<td>352</td>
<td>384</td>
<td>423</td>
<td>468</td>
</tr>
<tr>
<td>155 cm</td>
<td>315</td>
<td>329</td>
<td>349</td>
<td>375</td>
<td>407</td>
<td>446</td>
<td>491</td>
</tr>
<tr>
<td>160 cm</td>
<td>339</td>
<td>352</td>
<td>372</td>
<td>398</td>
<td>431</td>
<td>470</td>
<td>515</td>
</tr>
<tr>
<td>165 cm</td>
<td>363</td>
<td>377</td>
<td>396</td>
<td>423</td>
<td>455</td>
<td>494</td>
<td>539</td>
</tr>
<tr>
<td>170 cm</td>
<td>388</td>
<td>402</td>
<td>422</td>
<td>448</td>
<td>480</td>
<td>519</td>
<td>564</td>
</tr>
<tr>
<td>175 cm</td>
<td>414</td>
<td>428</td>
<td>447</td>
<td>474</td>
<td>506</td>
<td>545</td>
<td>590</td>
</tr>
<tr>
<td>180 cm</td>
<td>441</td>
<td>454</td>
<td>474</td>
<td>500</td>
<td>533</td>
<td>571</td>
<td>616</td>
</tr>
<tr>
<td>185 cm</td>
<td>468</td>
<td>481</td>
<td>501</td>
<td>527</td>
<td>560</td>
<td>599</td>
<td>644</td>
</tr>
</tbody>
</table>
### FEV₁ (L) Female Children (<18 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>1.45</td>
<td>1.58</td>
<td>1.71</td>
<td>1.84</td>
<td>1.97</td>
<td>2.10</td>
</tr>
<tr>
<td>130 cm</td>
<td>1.59</td>
<td>1.73</td>
<td>1.86</td>
<td>1.99</td>
<td>2.12</td>
<td>2.25</td>
</tr>
<tr>
<td>135 cm</td>
<td>1.75</td>
<td>1.88</td>
<td>2.01</td>
<td>2.14</td>
<td>2.27</td>
<td>2.40</td>
</tr>
<tr>
<td>140 cm</td>
<td>1.91</td>
<td>2.04</td>
<td>2.17</td>
<td>2.30</td>
<td>2.43</td>
<td>2.56</td>
</tr>
<tr>
<td>145 cm</td>
<td>2.07</td>
<td>2.20</td>
<td>2.33</td>
<td>2.46</td>
<td>2.59</td>
<td>2.72</td>
</tr>
<tr>
<td>150 cm</td>
<td>2.24</td>
<td>2.37</td>
<td>2.50</td>
<td>2.63</td>
<td>2.76</td>
<td>2.89</td>
</tr>
<tr>
<td>155 cm</td>
<td>2.41</td>
<td>2.54</td>
<td>2.68</td>
<td>2.81</td>
<td>2.94</td>
<td>3.07</td>
</tr>
<tr>
<td>160 cm</td>
<td>2.59</td>
<td>2.73</td>
<td>2.86</td>
<td>2.99</td>
<td>3.12</td>
<td>3.25</td>
</tr>
<tr>
<td>165 cm</td>
<td>2.78</td>
<td>2.91</td>
<td>3.04</td>
<td>3.17</td>
<td>3.30</td>
<td>3.44</td>
</tr>
<tr>
<td>170 cm</td>
<td>2.97</td>
<td>3.11</td>
<td>3.24</td>
<td>3.37</td>
<td>3.50</td>
<td>3.63</td>
</tr>
<tr>
<td>175 cm</td>
<td>3.17</td>
<td>3.30</td>
<td>3.43</td>
<td>3.56</td>
<td>3.70</td>
<td>3.83</td>
</tr>
<tr>
<td>180 cm</td>
<td>3.38</td>
<td>3.51</td>
<td>3.64</td>
<td>3.77</td>
<td>3.90</td>
<td>4.03</td>
</tr>
<tr>
<td>185 cm</td>
<td>3.59</td>
<td>3.72</td>
<td>3.85</td>
<td>3.98</td>
<td>4.11</td>
<td>4.24</td>
</tr>
</tbody>
</table>

### FVC (L) Female Children (<18 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>1.58</td>
<td>1.70</td>
<td>1.82</td>
<td>1.93</td>
<td>2.05</td>
<td>2.17</td>
</tr>
<tr>
<td>130 cm</td>
<td>1.77</td>
<td>1.89</td>
<td>2.01</td>
<td>2.12</td>
<td>2.24</td>
<td>2.36</td>
</tr>
<tr>
<td>135 cm</td>
<td>1.97</td>
<td>2.08</td>
<td>2.20</td>
<td>2.32</td>
<td>2.44</td>
<td>2.56</td>
</tr>
<tr>
<td>140 cm</td>
<td>2.17</td>
<td>2.29</td>
<td>2.41</td>
<td>2.52</td>
<td>2.64</td>
<td>2.76</td>
</tr>
<tr>
<td>145 cm</td>
<td>2.38</td>
<td>2.50</td>
<td>2.62</td>
<td>2.73</td>
<td>2.85</td>
<td>2.97</td>
</tr>
<tr>
<td>150 cm</td>
<td>2.60</td>
<td>2.72</td>
<td>2.84</td>
<td>2.95</td>
<td>3.07</td>
<td>3.19</td>
</tr>
<tr>
<td>155 cm</td>
<td>2.82</td>
<td>2.94</td>
<td>3.06</td>
<td>3.18</td>
<td>3.30</td>
<td>3.42</td>
</tr>
<tr>
<td>160 cm</td>
<td>3.06</td>
<td>3.18</td>
<td>3.29</td>
<td>3.41</td>
<td>3.53</td>
<td>3.65</td>
</tr>
<tr>
<td>165 cm</td>
<td>3.30</td>
<td>3.42</td>
<td>3.54</td>
<td>3.65</td>
<td>3.77</td>
<td>3.89</td>
</tr>
<tr>
<td>170 cm</td>
<td>3.55</td>
<td>3.66</td>
<td>3.78</td>
<td>3.90</td>
<td>4.02</td>
<td>4.14</td>
</tr>
<tr>
<td>175 cm</td>
<td>3.80</td>
<td>3.92</td>
<td>4.04</td>
<td>4.16</td>
<td>4.28</td>
<td>4.39</td>
</tr>
<tr>
<td>180 cm</td>
<td>4.07</td>
<td>4.18</td>
<td>4.30</td>
<td>4.42</td>
<td>4.54</td>
<td>4.66</td>
</tr>
<tr>
<td>185 cm</td>
<td>4.34</td>
<td>4.45</td>
<td>4.57</td>
<td>4.69</td>
<td>4.81</td>
<td>4.93</td>
</tr>
</tbody>
</table>
### FEV₁/FVC (%) Female Children (<18 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Heights</td>
<td>89.1</td>
<td>88.7</td>
<td>88.3</td>
<td>87.8</td>
<td>87.4</td>
<td>87.0</td>
</tr>
</tbody>
</table>

### PEF (L/min) Female Children (<18 years)

<table>
<thead>
<tr>
<th>Age</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
<th>16</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 cm</td>
<td>184</td>
<td>220</td>
<td>249</td>
<td>269</td>
<td>281</td>
<td>285</td>
</tr>
<tr>
<td>130 cm</td>
<td>198</td>
<td>235</td>
<td>263</td>
<td>283</td>
<td>295</td>
<td>299</td>
</tr>
<tr>
<td>135 cm</td>
<td>213</td>
<td>249</td>
<td>278</td>
<td>298</td>
<td>310</td>
<td>314</td>
</tr>
<tr>
<td>140 cm</td>
<td>228</td>
<td>265</td>
<td>293</td>
<td>313</td>
<td>325</td>
<td>329</td>
</tr>
<tr>
<td>145 cm</td>
<td>244</td>
<td>281</td>
<td>309</td>
<td>329</td>
<td>341</td>
<td>345</td>
</tr>
<tr>
<td>150 cm</td>
<td>261</td>
<td>297</td>
<td>325</td>
<td>346</td>
<td>358</td>
<td>362</td>
</tr>
<tr>
<td>155 cm</td>
<td>278</td>
<td>314</td>
<td>342</td>
<td>363</td>
<td>375</td>
<td>379</td>
</tr>
<tr>
<td>160 cm</td>
<td>295</td>
<td>332</td>
<td>360</td>
<td>380</td>
<td>392</td>
<td>396</td>
</tr>
<tr>
<td>165 cm</td>
<td>314</td>
<td>350</td>
<td>378</td>
<td>398</td>
<td>411</td>
<td>415</td>
</tr>
<tr>
<td>170 cm</td>
<td>332</td>
<td>369</td>
<td>397</td>
<td>417</td>
<td>429</td>
<td>433</td>
</tr>
<tr>
<td>175 cm</td>
<td>352</td>
<td>388</td>
<td>416</td>
<td>436</td>
<td>449</td>
<td>453</td>
</tr>
<tr>
<td>180 cm</td>
<td>371</td>
<td>408</td>
<td>436</td>
<td>456</td>
<td>468</td>
<td>472</td>
</tr>
<tr>
<td>185 cm</td>
<td>392</td>
<td>428</td>
<td>456</td>
<td>477</td>
<td>489</td>
<td>493</td>
</tr>
</tbody>
</table>
## Asthma Action Plan

**Name:** ........................................  **Date:** ........................................  **Best Peak Flow:** ........................................  
*Not recommended for children under 12 years*

### WHEN WELL

**Asthma under control (almost no symptoms)**

<table>
<thead>
<tr>
<th>Medicine Type</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preventer</td>
<td></td>
</tr>
<tr>
<td>Reliever</td>
<td></td>
</tr>
<tr>
<td>Symptom controller (if prescribed)</td>
<td></td>
</tr>
<tr>
<td>Combination medication (if prescribed)</td>
<td></td>
</tr>
</tbody>
</table>

### WHEN NOT WELL

**Asthma getting worse (waking from sleep, at the first sign of a cold, using more reliever)**

<table>
<thead>
<tr>
<th>Medicine Type</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preventer</td>
<td></td>
</tr>
<tr>
<td>Reliever</td>
<td></td>
</tr>
<tr>
<td>Continue symptom controller</td>
<td></td>
</tr>
<tr>
<td>Continue combination medication</td>
<td></td>
</tr>
</tbody>
</table>

**IF SYMPTOMS GET WORSE**

**Asthma is severe**

- **Start prednisolone/prednisone and contact doctor** Dose ........................................
- **Stay on this dose until your peak flow is above** on two consecutive mornings
- **Reduce prednisolone/prednisone to dose** daily for days, then cease

**Extra steps to take** .................................................................

**When your symptoms get better, return to the dose you take when well.**

### DANGER SIGNS

**Symptoms get worse very quickly, need reliever more than 2 hourly**

- **Continue reliever** .................................................................  
**Dial 000 for ambulance**

**Peak flow below**

---

**To order more Asthma Action Plans,**
**please call the National Asthma Council order line: 1800 032 495**

---

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
## Asthma Action Plan

### WHEN WELL

You will
- be free of regular night-time wheeze or cough or chest tightness
- have no regular wheeze or cough or chest tightness on waking or during the day
- be able to take part in normal physical activity without getting asthma symptoms
- need reliever medication less than 3 times a week (except if it is used before exercise)

### WHEN NOT WELL

You will
- have increasing night-time wheeze or cough or chest tightness
- have symptoms regularly in the morning when you wake up
- have a need for extra doses of reliever medication
- have symptoms which interfere with exercise
  (You may experience one or more of these)

### IF SYMPTOMS GET WORSE, THIS IS AN ACUTE ATTACK

You will
- have one or more of the following: wheeze, cough, chest tightness or shortness of breath
- need to use your reliever medication at least once every 3 hours or more often

### DANGER SIGNS

- your symptoms get worse very quickly
- wheeze or chest tightness or shortness of breath continue after reliever medication or return within minutes of taking reliever medication
- severe shortness of breath, inability to speak comfortably, blueness of lips

**IMMEDIATE ACTION IS NEEDED: CALL AN AMBULANCE**

Doctor's stamp and/or contact details:  
Pharmacist's stamp and/or contact details:

---

Take this Action Plan with you when you visit your doctor

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Self-management education checklist

These issues might be addressed when helping patients to become proficient at managing their asthma. Whether and when each of these is included would depend on the patient’s priorities and willingness to carry out day-to-day self-management practices. Use illustrations to explain these concepts. Remember to use plain English.

What is asthma?
- Asthma is an inflammatory disease
- The underlying condition or tendency remains even when symptoms are absent
- In people with asthma, the airways narrow and don’t function normally when exposed to a trigger factor
- Triggers are sometimes difficult to identify
- During an asthma episode, a combination of factors causes airway narrowing:
  - Smooth muscle spasm
  - Airway swelling, due to:
    - oedema – fluid and proteins deposited across the airway wall
    - mucus hypersecretion
    - muscle and mucous gland enlargement.

How asthma is treated
Action and role of each of the classes of asthma medications:
- Reliever medication (bronchodilators)
- Preventer medication (anti-inflammatory agents)
- Symptom controllers (long-acting beta² agonists)
- Combination medications (preventer plus symptom controller)

Emphasise the important role of preventers in those who need them.
- The difference between preventers and relievers
- Preventers must be taken regularly, irrespective of symptoms
- Common side-effects and how to cope with these

Delivery devices
- Care of the device
- Correct inhaler technique (demonstrate and practice until patient can perform correctly)

Key facts about childhood asthma
- Childhood asthma is common: 30% of children will have a form of asthma at some stage.
- Infrequent virus-induced wheezing in infancy improves by age 6 years in most children.
- Allergy is an important cause of asthma in children and can trigger acute attacks of asthma. Continuing asthma is more likely if eczema and hay fever are also present and there is ongoing allergen exposure.
- More than half the children with mild asthma will be free of symptoms or have only mild intermittent wheezing in later life.
- Moderate or severe asthma rarely goes away by itself, even in adolescents.
- Stopping treatment results in a return of symptoms, usually within days to weeks.

Negotiate a plan of care and review and monitor the plan
- Reinforce the need for long-term adherence to preventive therapy.

What to expect about the duration of treatment:
- Asthma treatment is usually long-term
- Beginning treatment with asthma medications does not necessarily mean that life-long treatment will be necessary
- Discourage the notion that treatment can be discontinued as soon as the symptoms resolve
- Emphasise the importance of attending for regular review of the current management plan.

Monitor and manage the symptoms and signs of asthma
Develop a written asthma action plan:
- How to recognise deteriorating asthma (peak expiratory flow monitoring or symptoms)
- Steps to take if asthma control deteriorates, including:
  - when to increase medication and by how much
  - when and how to seek medical treatment
- If using peak expiratory flow measurement to monitor asthma:
  - Instruct in correct technique and maintenance
  - Tailor the monitoring schedule to the person’s daily program

How to avoid asthma exacerbations
- Recognising and avoiding triggers
- Smoking cessation and avoiding other people’s smoke
- Managing exercise-induced asthma

Minimising the effects of asthma on everyday life
Identify aspects of daily life (work, school, social activities) that are affected by asthma and develop strategies for minimising effects:
- Effects of asthma on physical function
- Effects on emotions
- Effects on interpersonal relationships.
First Aid for Asthma

WHAT IS AN ASTHMA ATTACK?

People with asthma have extra-sensitive airways. Triggers like dust, pollens, animals, tobacco smoke and exercise may make their airways swell and narrow, causing wheeze, cough and difficulty breathing.

1. Sit the person comfortably upright. Be calm and reassuring.

2. Give 4 puffs of a blue Reliever inhaler (puffer) – Ventolin, Airomir, Bricanyl, or Asmol. Relievers are best given through a spacer, if available. Use 1 puff at a time and ask the person to take 4 breaths from the spacer after each puff. Use the person's own inhaler if possible. If not, use the First Aid Kit inhaler or borrow one from someone.

3. Wait 4 minutes. If there is no improvement, give another 4 puffs.

4. If little or no improvement, CALL AN AMBULANCE IMMEDIATELY (DIAL 000) and state that the person is having an asthma attack. Keep giving 4 puffs every 4 minutes until the ambulance arrives.

   Children: 4 puffs each time is a safe dose.
   Adults: up to 6-8 puff every 5 minutes may be given for a severe attack while waiting for the ambulance.

WITH SPACER

- Shake inhaler and insert mouthpiece into spacer.
- Place spacer mouthpiece in person's mouth and fire 1 puff.
- Ask the person to breathe in and out normally for about 4 breaths.
- Repeat in quick succession until 4 puffs have been given.

WITHOUT SPACER

- Shake inhaler
- Place mouthpiece in person’s mouth. Fire 1 puff as the person inhales slowly and steadily.
- Ask the person to hold that breath for 4 seconds, then take 4 normal breaths.
- Repeat until 4 puffs have been given.

WHAT IF IT IS THE FIRST ATTACK OF ASTHMA?

If someone collapses and appears to have difficulty breathing, CALL AN AMBULANCE IMMEDIATELY whether or not the person is known to have asthma.

- Give four puffs of a Reliever and repeat if no improvement.
- Keep giving 4 puffs every 4 minutes until the ambulance arrives.

No harm is likely to result from giving a Reliever to someone who does not have asthma.

For more information on asthma, contact your local Asthma Foundation 1800 645 130

For more copies of this chart, contact the National Asthma Council 1800 032 495

Although all care has been taken, this chart is a general guide only and is not intended to be a substitute for individual medical advice/treatment. The National Asthma Council Australia expressly disclaims all responsibility (including for negligence) for any loss, damage or personal injury resulting from reliance on the information contained herein.

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Glossary of Asthma Terms

Asthma plans, tools and resources

Asthma control tools
Validated questionnaires to assess a patient’s level of asthma control. They are designed to assist the GP with basic information for monitoring of asthma, and to educate the patient on their level of asthma control.

Asthma Management Plan
A name derived from the 1989 Medical Journal of Australia article by The Thoracic Society of Australia and New Zealand, the Asthma Management Plan, i.e. Australia’s consensus treatment guidelines for asthma – the first in the world.

Originally the name of what is now the Asthma Management Handbook.

Six Step Asthma Management Plan
The Asthma Management Plan is based on the Six Step Asthma Management Plan:
1. Assess asthma severity
2. Achieve best lung function
3. Maintain best lung function: identify and avoid trigger factors
4. Maintain best lung function: optimise medication program
5. Develop an action plan
6. Educate and review regularly

Sometimes referred to as the Australian Six Step Asthma Management Plan. The components of the Six Step Plan are covered in Ongoing care in this publication.

(Written) Asthma Action Plan
Usually a proforma (there are a variety of formats), which the doctor completes with the patient, who then uses it to manage their asthma on a day-to-day basis.

Also referred to as asthma management plan (confusing), asthma plan, self-management plan, asthma care plan, personal asthma plan.

Asthma Cycle of Care
The 3+ Visit Plan developed by the National Asthma Council Australia’s GP Asthma Group has been replaced by the Asthma Cycle of Care, also for moderate to severe asthma. These changes to the Australian Government’s GP Initiative were introduced in response to feedback mainly from GPs, on how it could be improved for use in general practice.

The Asthma Cycle of Care involves at least two visits to a GP over a period of 12 months. These visits will include:

- diagnosis and assessment of asthma severity and level of control;
- development of a written asthma action plan;
- provision of information and patient self-management education; and
- review of asthma management and asthma action plan.


Care Plan
Part of the Enhanced Primary Care Medicare items – care plans are comprehensive, longitudinal plans for the care of the individual patient. They are available for people of any age with chronic conditions (or one which will last for 6 months or more or is terminal).

They require the involvement of the patient’s usual general practitioner and two other health professionals (who must provide different types of services).

Care plans provide the opportunity for the patient’s GP to work with other health professionals and care providers to develop, review or contribute to care plans for people with one or more chronic conditions and multidisciplinary care needs. Care plans may be developed for people who are in the community (community care plans) or who are being discharged back into the community (discharge care plans).

Emergency Management of Asthma Chart
National Asthma Council wall chart for hospital Emergency Departments.
4 x 4 x 4 Plan, Asthma Emergency Plan, Asthma First Aid, First Aid for Asthma Chart

Names used to describe the various first aid charts of the National Asthma Council and Asthma Foundations, which use the 4 puffs x 4 breaths x 4 minutes guidelines set by The Thoracic Society of Australia and New Zealand. These are to provide first aiders and/or the public with advice on what to do if someone develops serious asthma symptoms.

Student Asthma Record

Asthma Friendly Schools program – students must provide the school with information about their asthma e.g. written asthma action plan, personal details, personal first aid plan.

Terminology

Anaphylaxis

Anaphylaxis, or anaphylactic reactions, are sudden, widespread, potentially severe and life-threatening allergic reactions. An anaphylactic reaction does not usually occur after the first exposure to an allergen but may occur after a subsequent exposure.

Anticholinergics

Agents that inhibit the cholinergic (parasympathetic) system.

Atopy

A personal or familial tendency to produce IgE antibodies to low doses of allergens, and, as a consequence, to develop typical symptoms such as asthma, rhinitis/conjunctivitis or atopic eczema/dermatitis.

Back-titrate

To determine the optimum dose of medication following introduction of a high dose by means of a gradual, stepwise reduction and observation of effect.

Bronchial hyperresponsiveness (also termed bronchial hyperreactivity)

Tendency of the smooth muscle of the tracheobronchial tree to contract more intensely in response to a given stimulus than it does in normal individuals. This condition is present in virtually all symptomatic patients with asthma. The most prominent manifestation of this smooth muscle contraction is a decrease in airway calibre that can be readily measured by lung function testing.

Bronchiectasis

Chronic dilation of the bronchi (the larger air passages in the lungs), marked by daily cough and sputum production.

Bronchiolitis

A contagious viral infection of the airways of infants and young children that causes difficulty in breathing, especially expiration.

Bronchodilator

A medication that acts to dilate (enlarge) the lumen of the airway to allow the unrestricted passage of air. These medications are commonly given to those with asthma who manifest wheezing.

Chronic bronchitis

Chronic bronchitis is a characterised by a cough that produces sputum for 3 months or more during 2 successive years; the cough is not due to another lung disease.

Combination medications

A combination of an inhaled corticosteroid and a long-acting beta-agonist in one device.

Corticosteroids

A group of synthetic hormones including prednisone, prednisolone, methylprednisolone and used in the treatment of asthma. See Preventers.

Cromones (also termed cromolyns)

Medications that can help to reduce allergen-induced responses following short-term exposure. See Preventers.

COPD

Chronic obstructive pulmonary disease. Also known as COAD (A for airways) or CORD (R for respiratory), i.e. chronic bronchitis, emphysema.

DPI

Dry-powder inhaler

Emphysema

Irreversible elargement of many of the 300 million air sacs (alveoli) that make up the lungs and destruction of the alveolar walls.
**FEV₁**
Forced expiratory volume in one second; the volume expired in the first second of maximal expiration after a maximal inspiration and a useful measure of how quickly full lungs can be emptied.

**ICS**
Inhaled corticosteroid medication. See Preventers.

**Immunotherapy**
Any form of treatment that uses the body’s natural abilities that constitute the immune system to fight infection and disease or to protect the body from some of the adverse effects of treatment. In this publication, the specific immunotherapy referred to is desensitisation, which is stimulation of the immune system with gradually increasing doses of the substances to which a person is allergic, the aim being to modify or stop the allergy “war” (by reducing the strength of the IgE and its effect on the mast cells). Allergy immunotherapy usually takes 6 months to a year to be effective and injections are usually required for 3–5 years.

**Leukotriene receptor antagonists (LTRAs)**
Leukotriene receptor antagonists are oral medications that specifically inhibit the production or actions of the inflammatory mediators in asthma (leukotrienes C4 and D4). See Preventers.

**Long-acting beta₂ agonists (LABAs)**
Bronchodilators used for prevention /control of inflammation rather than for acute symptoms of asthma, except in the case of the LABA eformoterol, which can be used as a reliever medication due to its rapid onset of action. See Symptom Controllers.

**MDI**
Metered-dose inhaler

**Nebulisation**
Conversion to a spray/mist by compressed air through a jet nebuliser

**NSAID**
Nonsteroidal anti-inflammatory drug (e.g. aspirin and other non-opioid analgesics) used for arthritis and other inflammatory disorders. Some people with asthma may have an asthma exacerbation as a reaction to NSAIDs.

**PEF (also termed PEFR)**
Peak expiratory flow (rate), measured with a peak flow meter.

**Preventers**
Preventer medications are those for long-term control of asthma and airway inflammation, e.g. ICS, cromones, leukotriene antagonists. Oral corticosteroids are ‘rescue’ medication for acute exacerbations and are not usually taken long-term.

**RAST testing**
Radioallergosorbent testing: to measure specific IgE antibodies in serum. Used as an alternative to skin tests to determine sensitivity to specific allergens.

**Relievers**
Reliever medications are those designed for rapid relief of symptoms, e.g short-acting beta₂ agonists (SABAs): salbutamol, terbutaline.

**Reversible airflow limitation (also termed airway obstruction)**
Where a 12% or greater improvement in FEV₁, 10-15 minutes after 400 mg salbutamol is administered, is achieved.

**Short-acting beta₂ agonists (SABAs)**
Bronchodilators for rapid relief of asthma symptoms. See Relievers.

**Spirometry**
Measuring the air entering and leaving the lungs by means of a spirometer.

**Symptom controllers**
Symptom controllers are those medications designed for long-term control /relief of symptoms, e.g. long-acting beta₂ agonists (LABAs). These drugs are given in addition to an ICS and are not for ‘stand-alone’ use.

**Wheezing**
Breathing with difficulty, usually with a whistling sound.
Aboriginal and Torres Strait Islander patients 81
Accuhaler, as drug delivery device 35
Accuhaler (fluticasone) 29
Accuhaler (Seretide) 33
acetylsalicylic acid 55
action plans see asthma action plans
acupuncture 53
acute asthma, beta2 agonist delivery for 37
acute asthma management in adults 38–42
follow-up care 41
history 40
initial assessment 38–39
other investigations 41
steps in emergency care 39–40
treatment 40, 42
  give SABA via MDI plus spacer immediately 40
  role of other agents 41
  start systemic corticosteroids 40
acute asthma management in children 43–6
community-based first aid 45
follow-up care 45
initial assessment 43, 44
steps in emergency care 43
treatment 43, 46
  mild acute asthma episode 44
  moderate acute asthma episode 44
  severe acute asthma episode 43
adherence to reliever medication/preventer medication 77–8
adolescents see asthma in adolescents
adrenaline 40, 62
adult-onset asthma, questions to ask patients 99
adults see acute asthma management in adults; asthma in adults
Aerolizer (Foradile) 32
Aged Care Health Assessments 81
aged patients see elderly
air environment 72
air pollutants 72
Airomir 25
airway obstruction 4
allergen avoidance
  and reduced asthma risk 118–19
  in children 120
allergen–gene vaccination 63
allergen–peptide immunotherapy 63
allergen-specific IgE 59, 100
allergens, inhalant
  as triggers for asthma 58–9
  strategies to reduce exposure to 58
allergic conjunctivitis 61, 120
allergic rhinitis
  allergen avoidance 60
  and asthma 57, 60–1
diagnosis 60
differential diagnosis 60
drug therapy 60–1
immunotherapy (desensitisation) 61
in adults 6
specialist referral for 61
allergic triggers 58–9, 71–2, 118–19
allergy
  and asthma 57–63
  food 58, 59
allergy skin prick tests (SPT) 56, 59, 100
allergy specialists, referral to 61, 71
allergy tests 8, 59–60, 100
alternative diagnostic tests 54
Alvesco 29
aminophylline 40
antenatal care 102
anti-Fce, use in asthma 63
anti-IgE monoclonal antibody therapy 31, 63
dosage 31
indications 31
anti-immunoglobulin therapy 31, 63
anti-inflammatories 4
antibiotics 34
anticholinergic agents, for COPD 92
anticholinergic sprays, for rhinorrhoea 61
antihistamines 34
  for allergic rhinitis 60
antioxidants 56, 63
anxiety disorders
  and asthma 115
  in children with asthma 115
Apoven 250 26
aromatherapy 54
Asmol 25
aspirin 71
assessment of asthma
  in adults and adolescents 68
  objective lung function tests 68
  in children 68–9
  objective lung function tests 69
  physical examination 69
  symptoms 68–9
assessment of asthma control 66
  questions to ask at every consultation 66
  versus severity 67
assessment of severity 67
  versus control 67
Asthma 3+ Visit Plan 79, 131
asthma
  and allergic rhinitis 57, 60–1
  and allergy 57–63
  and anxiety disorders 115
  and COPD 89, 90
and depression 114
and diet 55–6
and mental illness 114–15
and pregnancy 101–3
and smoking 82–7, 120
definition 4
distinguishing from COPD 107
in Australia, basic facts 3
in the elderly 81, 104–11
not a psychosomatic illness 114
asthma action plans 74–5, 127–8
for adults 75
for children and adolescents 75
for elderly 111
asthma clinics 79
asthma control 65–70
assessment of asthma
in adults and adolescents 69
in children 68–9
assessment of control 66
assessment of severity 67
asthma history checklist for new patients 66
identify patients with high-risk asthma 70
ongoing review 70
severity versus control 67
Asthma Control Questionnaire 66
asthma control tools 66, 131
asthma education see self-management education
Asthma Emergency Plan 32
asthma exacerbations see exacerbations
Asthma First Aid 32
asthma history checklist for new patients 66
asthma in adolescents
assessment 68
asthma action plans 75
drug treatment principles 22–4
self-management 75
asthma in adults
adjusting maintenance therapy 18–19
assessment 68
asthma action plans 75
classification 8–10
detection and diagnosis 5–8
diagnostic testing 6–8
drug treatment principles 15–16, 17–23
examination 6
history 5–6
review regularly for optimum control 18
review the history 77
see also acute asthma management in adults
asthma in children
and anxiety disorders 115
assessment 68–9
asthma action plans 75
classification 13–14
delivery devices 36–7
diagnoses confused with asthma 12
diagnosis 11–12
diagnostic testing 10, 69
drug treatment principles 16, 22–4
patterns of 12–13
prevention therapy 24
review the history 77
stepwise approach to drug therapy 23
symptoms 11, 68–9
asthma management, organising practice for 78–81
Asthma Management Plan 1, 131
review 80
asthma medications 4
combination medications 15, 16, 17, 19, 22, 29, 33–4
preventioners 15, 16, 24, 27–32
relievers 15, 16, 17, 25–7
symptom controllers 32
use in competitive sport 97
see also specific medications
asthma prevention 116–20
primary prevention 117–19
secondary prevention 120
tertiary prevention 120
asthma review, organisation of 70, 79
Asthma Score 66
atopic dermatitis 120
atopy 83, 84
atrophic rhinitis 61
Atovent 26
attitude of patients 77
Aust L codes 52
Aust R codes 52
Australian Sports Anti-Doping Authority 97
Austyn 26
Autohaler (BDP-HFA) 29
Autohaler (salbutamol) 26
back-titrating combination therapy 21–2
bacterial endotoxins 119
BDP-HFA 27, 28–9
dosage 29
ICS dose level 10
beclomethasone dipropionate see BDP-HFA
beta-adrenergic blocking agents 71
beta2 agonist delivery
for acute asthma 37
for stable asthma 37
bioelectric tests 60
blood assays for essential fatty acids, vitamins and minerals 45
blue inhalers 26
breastfeeding 56, 103, 117–18
breathing techniques 52–3
breathlessness in children 69
Bricanyl 25
bronchial hyperresponsiveness 83
bronchiectasis 12
bronchitis 12, 89
bronchoconstriction
exercise-induced see exercise-induced asthma
from food protein allergy 55
predictable 35
bronchodilators 4, 16
for wheezing in children 23
Brondecon elixir 27
brown inhaler 27
Bryan’s test 54
BUD 27, 29
dosage 29
for exacerbations 49
ICS dose level 10
in pregnancy 101
budesonide see BUD
budesonide plus eformoterol 15, 29, 33, 34
dosage 34
for exacerbations 49
dose adjustment 22
onset of therapeutic action 16, 19, 33
presentation 34
use as reliever 17
bupropion sustained release 85
bushfire smoke 72
Buteyko breathing technique 53
CAM see complementary and alternative medicine in asthma
camomile 52
carbachol 71
Care Plan 131
caregivers’ mental health 115
cat allergens 58
cetirizine 120
challenge tests 8
Chemart Ipratropium 26
chest X-rays 9, 40
children see acute asthma management in children;
asthma in children
chiropractic manipulation 53
choline theophyllinate 27
cholinergic agents 71
cholinesterase inhibitors 71
chronic bronchitis 12
chronic obstructive pulmonary disease see COPD
chronic suppurative lung disease 12
CIC 27, 29
dosage 29
ICS dose level 10
ciclesonide see CIC
cigarette smoking see smoking
classification
asthma in adults 8–10
untreated asthma 8–10
asthma in children 13–14
cockroaches 58
combination devices 37
combination medications 33–4
budesonide plus eformoterol 15, 16, 17, 19, 29, 33, 34
fluticasone plus salmeterol 16, 19, 22, 29, 33
community-based first aid 44
community pharmacist, role of 80–1
comorbidities 91, 112–15
competitive sport, asthma medications in 97
complementary and alternative medicine in asthma 51–2
acupuncture 53
breathing techniques 52–3
clinical evaluation and regulation 52
dietary modifications 53
exercise therapies 53
information resources 54
manual therapies 53
medicinal therapies 53–4
other therapies 54
potential adverse effects 52
psychological therapies 54
Comprehensive Medical Assessments 81
continuous positive airway pressure (CPAP) 113
control see asthma control
COPD 26, 88–92
and asthma 89, 90
comorbidities 91
diagnosis 90
distinguishing from asthma 107
FEV1/FVC in 91, 107
history 90
in elderly 91, 105, 106, 107
management 92–3
differences from asthma management 92
drug treatment 92
home oxygen therapy 92
overlap with bronchitis, emphysema and asthma 89
physical examination 90
prevalence 89
risk factors 89–90
spirometry 91, 107–8
sputum or breath condensate biomarkers 91
symptoms 90
COPDX guidelines 91
corticosteroids
inhaled see ICS therapy
oral 32, 39, 48, 49, 61, 92
parenteral 39
systemic 39
cough
due to asthma 12
in children 68
recurrent non-specific, in children 12
‘cough-variant asthma’ syndrome 12
cows’ milk formula, in infants 56, 118
cranial therapy 53
cromones 27, 30–1
culturally and linguistically diverse groups 81
cytokine therapy 63
cytotoxic food testing 54
<table>
<thead>
<tr>
<th>DBL Ipratropium 26</th>
<th>DBL Ipratropium 26</th>
</tr>
</thead>
<tbody>
<tr>
<td>decongestants</td>
<td>decongestants</td>
</tr>
<tr>
<td>intranasal, for allergic rhinitis 60–1</td>
<td>oral, for allergic rhinitis 61</td>
</tr>
<tr>
<td>delivery devices 35–7</td>
<td>delivery devices 35–7</td>
</tr>
<tr>
<td>deposition 35</td>
<td>deposition 35</td>
</tr>
<tr>
<td>device training 36, 74</td>
<td>device training 36, 74</td>
</tr>
<tr>
<td>for children 36–7</td>
<td>for children 36–7</td>
</tr>
<tr>
<td>prescribing devices 35</td>
<td>prescribing devices 35</td>
</tr>
<tr>
<td>use and care of spacers 36</td>
<td>use and care of spacers 36</td>
</tr>
<tr>
<td>depression, and asthma 114</td>
<td>depression, and asthma 114</td>
</tr>
<tr>
<td>desensitisation, for allergic rhinitis 61</td>
<td>desensitisation, for allergic rhinitis 61</td>
</tr>
<tr>
<td>device training 36, 74</td>
<td>device training 36, 74</td>
</tr>
<tr>
<td>diabetes, and asthma 109</td>
<td>diabetes, and asthma 109</td>
</tr>
<tr>
<td>diagnosis</td>
<td>diagnosis</td>
</tr>
<tr>
<td>asthma in adults 6–8</td>
<td>asthma in adults 6–8</td>
</tr>
<tr>
<td>asthma in children 11–12</td>
<td>asthma in children 11–12</td>
</tr>
<tr>
<td>asthma in elderly 105</td>
<td>asthma in elderly 105</td>
</tr>
<tr>
<td>COPD 90</td>
<td>COPD 90</td>
</tr>
<tr>
<td>occupational asthma 99–100</td>
<td>occupational asthma 99–100</td>
</tr>
<tr>
<td>diagnostic tests</td>
<td>diagnostic tests</td>
</tr>
<tr>
<td>alternative 54</td>
<td>alternative 54</td>
</tr>
<tr>
<td>in adults 6–8</td>
<td>in adults 6–8</td>
</tr>
<tr>
<td>in children 11</td>
<td>in children 11</td>
</tr>
<tr>
<td>diet and asthma 55–6, 118</td>
<td>diet and asthma 55–6, 118</td>
</tr>
<tr>
<td>dietary allergen avoidance</td>
<td>dietary allergen avoidance</td>
</tr>
<tr>
<td>in infancy 118</td>
<td>in infancy 118</td>
</tr>
<tr>
<td>in pregnancy or lactation 118</td>
<td>in pregnancy or lactation 118</td>
</tr>
<tr>
<td>dietary modifications 53</td>
<td>dietary modifications 53</td>
</tr>
<tr>
<td>dose adjustment</td>
<td>dose adjustment</td>
</tr>
<tr>
<td>with budesonide plus eformoterol 22</td>
<td>with budesonide plus eformoterol 22</td>
</tr>
<tr>
<td>with fluticasone plus salmeterol 22</td>
<td>with fluticasone plus salmeterol 22</td>
</tr>
<tr>
<td>dowsing 54</td>
<td>dowsing 54</td>
</tr>
<tr>
<td>drug therapy, aim of 17</td>
<td>drug therapy, aim of 17</td>
</tr>
<tr>
<td>drug treatment principles 17</td>
<td>drug treatment principles 17</td>
</tr>
<tr>
<td>in adults 15–16, 17–23</td>
<td>in adults 15–16, 17–23</td>
</tr>
<tr>
<td>in children and adolescents 16, 22–4</td>
<td>in children and adolescents 16, 22–4</td>
</tr>
<tr>
<td>drugs see asthma medications; medications</td>
<td>drugs see asthma medications; medications</td>
</tr>
<tr>
<td>dry-powder inhaler (DPI) 35</td>
<td>dry-powder inhaler (DPI) 35</td>
</tr>
<tr>
<td>dust mites 58, 59, 71, 118, 120</td>
<td>dust mites 58, 59, 71, 118, 120</td>
</tr>
<tr>
<td>‘dysbiosis’ tests 54</td>
<td>‘dysbiosis’ tests 54</td>
</tr>
<tr>
<td>dyspnoea 12</td>
<td>dyspnoea 12</td>
</tr>
<tr>
<td>echinacea 52, 71</td>
<td>echinacea 52, 71</td>
</tr>
<tr>
<td>education see self-management education</td>
<td>education see self-management education</td>
</tr>
<tr>
<td>eformoterol 15, 26, 32</td>
<td>eformoterol 15, 26, 32</td>
</tr>
<tr>
<td>dosage 32</td>
<td>dosage 32</td>
</tr>
<tr>
<td>during pregnancy 103</td>
<td>during pregnancy 103</td>
</tr>
<tr>
<td>for COPD 92</td>
<td>for COPD 92</td>
</tr>
<tr>
<td>see also budesonide plus eformoterol</td>
<td>see also budesonide plus eformoterol</td>
</tr>
<tr>
<td>eggs 55</td>
<td>eggs 55</td>
</tr>
<tr>
<td>elderly</td>
<td>elderly</td>
</tr>
<tr>
<td>COPD in 91, 105</td>
<td>COPD in 91, 105</td>
</tr>
<tr>
<td>distinguishing COPD from asthma 107</td>
<td>distinguishing COPD from asthma 107</td>
</tr>
<tr>
<td>excluding diagnoses other than asthma and COPD 106</td>
<td>excluding diagnoses other than asthma and COPD 106</td>
</tr>
<tr>
<td>spirometry in 107–8</td>
<td>spirometry in 107–8</td>
</tr>
<tr>
<td>elderly and asthma 81, 104–11</td>
<td>elderly and asthma 81, 104–11</td>
</tr>
<tr>
<td>diagnosis 105</td>
<td>diagnosis 105</td>
</tr>
<tr>
<td>diagnostic difficulties 105</td>
<td>diagnostic difficulties 105</td>
</tr>
<tr>
<td>diagnostic steps 105</td>
<td>diagnostic steps 105</td>
</tr>
<tr>
<td>distinguishing asthma from COPD 107</td>
<td>distinguishing asthma from COPD 107</td>
</tr>
<tr>
<td>exacerbations and action plans 111</td>
<td>exacerbations and action plans 111</td>
</tr>
<tr>
<td>identifying patients with airflow limitation 105–6</td>
<td>identifying patients with airflow limitation 105–6</td>
</tr>
<tr>
<td>allergy 106</td>
<td>allergy 106</td>
</tr>
<tr>
<td>smoking history 106</td>
<td>smoking history 106</td>
</tr>
<tr>
<td>symptoms 105–6</td>
<td>symptoms 105–6</td>
</tr>
<tr>
<td>useful questions 106</td>
<td>useful questions 106</td>
</tr>
<tr>
<td>management 108–11</td>
<td>management 108–11</td>
</tr>
<tr>
<td>drug treatment 108–9</td>
<td>drug treatment 108–9</td>
</tr>
<tr>
<td>in diabetics 109</td>
<td>in diabetics 109</td>
</tr>
<tr>
<td>patient education 109</td>
<td>patient education 109</td>
</tr>
<tr>
<td>prescribing issues 109</td>
<td>prescribing issues 109</td>
</tr>
<tr>
<td>review 109–10</td>
<td>review 109–10</td>
</tr>
<tr>
<td>perception of airflow limitation 110</td>
<td>perception of airflow limitation 110</td>
</tr>
<tr>
<td>vaccination status 110–11</td>
<td>vaccination status 110–11</td>
</tr>
<tr>
<td>role of diagnostic treatment trial 108</td>
<td>role of diagnostic treatment trial 108</td>
</tr>
<tr>
<td>underdiagnosis 104–5</td>
<td>underdiagnosis 104–5</td>
</tr>
<tr>
<td>electrodermal testing 54</td>
<td>electrodermal testing 54</td>
</tr>
<tr>
<td>emergency care</td>
<td>emergency care</td>
</tr>
<tr>
<td>of adult with acute asthma 41–2</td>
<td>of adult with acute asthma 41–2</td>
</tr>
<tr>
<td>of child with acute asthma 45–6</td>
<td>of child with acute asthma 45–6</td>
</tr>
<tr>
<td>Emergency Management of Asthma Chart 131</td>
<td>Emergency Management of Asthma Chart 131</td>
</tr>
<tr>
<td>emphysema 89</td>
<td>emphysema 89</td>
</tr>
<tr>
<td>ENT specialists, referral to 61</td>
<td>ENT specialists, referral to 61</td>
</tr>
<tr>
<td>environmental tobacco smoke 72</td>
<td>environmental tobacco smoke 72</td>
</tr>
<tr>
<td>and increased asthma risk 117</td>
<td>and increased asthma risk 117</td>
</tr>
<tr>
<td>avoidance of in children 120</td>
<td>avoidance of in children 120</td>
</tr>
<tr>
<td>effects of exposure in people with asthma 83</td>
<td>effects of exposure in people with asthma 83</td>
</tr>
<tr>
<td>exposure to among children 83</td>
<td>exposure to among children 83</td>
</tr>
<tr>
<td>in utero exposure 84, 102, 103</td>
<td>in utero exposure 84, 102, 103</td>
</tr>
<tr>
<td>enzyme-potentiated immunotherapy 54</td>
<td>enzyme-potentiated immunotherapy 54</td>
</tr>
<tr>
<td>Epaq 25</td>
<td>Epaq 25</td>
</tr>
<tr>
<td>ethnic groups 81</td>
<td>ethnic groups 81</td>
</tr>
<tr>
<td>eucapnic voluntary hyperpnea 95</td>
<td>eucapnic voluntary hyperpnea 95</td>
</tr>
<tr>
<td>exacerbations 34</td>
<td>exacerbations 34</td>
</tr>
<tr>
<td>distinguishing from poor asthma control 48</td>
<td>distinguishing from poor asthma control 48</td>
</tr>
<tr>
<td>during pregnancy 102</td>
<td>during pregnancy 102</td>
</tr>
<tr>
<td>in the elderly 111</td>
<td>in the elderly 111</td>
</tr>
<tr>
<td>infections 72–3</td>
<td>infections 72–3</td>
</tr>
<tr>
<td>managing 32, 47–50</td>
<td>managing 32, 47–50</td>
</tr>
<tr>
<td>medications that exacerbate asthma 34–5, 71, 76–7</td>
<td>medications that exacerbate asthma 34–5, 71, 76–7</td>
</tr>
<tr>
<td>preventing 72–3</td>
<td>preventing 72–3</td>
</tr>
<tr>
<td>role of PEF monitoring in detecting 48</td>
<td>role of PEF monitoring in detecting 48</td>
</tr>
<tr>
<td>vaccinations 72–3</td>
<td>vaccinations 72–3</td>
</tr>
<tr>
<td>exacerbations in adults, managing 48–9</td>
<td>exacerbations in adults, managing 48–9</td>
</tr>
<tr>
<td>inhaled corticosteroids 49</td>
<td>inhaled corticosteroids 49</td>
</tr>
<tr>
<td>oral corticosteroids 48</td>
<td>oral corticosteroids 48</td>
</tr>
<tr>
<td>SABAs 48–9</td>
<td>SABAs 48–9</td>
</tr>
<tr>
<td>exacerbations in children, managing 49–50</td>
<td>exacerbations in children, managing 49–50</td>
</tr>
<tr>
<td>inhaled corticosteroids 50</td>
<td>inhaled corticosteroids 50</td>
</tr>
<tr>
<td>LTRAs 50</td>
<td>LTRAs 50</td>
</tr>
<tr>
<td>oral corticosteroids 50</td>
<td>oral corticosteroids 50</td>
</tr>
</tbody>
</table>
SABAs 50
exercise-induced asthma 93–7
and asthma medications in competitive sport 97
assessment of lung function 94–5
definition 93
detection 94
drug-free strategies 96
effect of training 95
impact on quality of life, asthma and sporting
performance 94
pathogenesis 93–4
treatment strategies 95–6
under- and over-diagnosis 95
exercise-induced bronchoconstriction see exercise-induced asthma
exercise-induced dyspnoea 12
exercise-induced laryngeal dysfunction 12
exercise-induced respiratory symptoms, in children 12
exercise therapies 53
exhaled nitric oxide test 8

FEV1
female adults 122
female children (<18 years) 125
in exercise-induced asthma 93, 94
in spirometry 7
male adults 121
male children (<20 years) 123

FEV1/FVC
female adults 122
female children (<18 years) 126
in asthma 107
in mild COPD 91, 108
male adults 121
male children (<20 years) 123
first aid for asthma 44, 130
First Aid for Asthma Chart 132
fish oil supplements 53, 118
fixed-dose ICS-LABA combination therapy regimens 21–2, 32
add-on therapy options 22
assessing asthma control 21
back-titrating combination therapy 21–2
dose adjustment with fluticasone plus salmeterol 22
gaining control 21
general principles using a combination inhaler 21
maintenance-and-reliever regimen 22
standard maintenance regimen 22
Flixotide 27, 29
fluticasone plus salmeterol 29, 33
dosage 32
for exacerbations 49
dose adjustment 22
onset of therapeutic action 16, 19, 33
fluticasone propionate see FP
follow-up care after an acute asthma episode
in adults 40
in children 44

food allergy 55
and asthma 59
food chemical intolerance 55, 72
food supplements 53, 63
foods
as asthma triggers 72
as potential treatment for asthma 56
Foradil 32
forced expiratory volume in 1 second see FEV1
forced vital capacity see FVC
formaldehyde 72
4 x 4 x 4 plan 44, 132
FP 27, 29
dosage 29
for exacerbations 49
ICS dose level 10
frequent intermittent asthma, in children 13, 14
FVC 6
female adults 122
female children (<18 years) 125
male adults 121
male children (<20 years) 123
see also FEV1/FVC
gastro-oesophageal reflux disease (GORD) 72, 113–14
and obesity 114
co-occurrence with OSA 113–14
treatment considerations in asthma patients 114
GenRx Ipratropium 26
Ginkgo biloba 52
glossary 132–3
grass pollen 58, 59, 120
green inhalers 25
H1-antihistamines, pre-treatment 62
hair analysis 54
Health Assessment patients, special considerations 81
Healthsense Ipratropium 26
heart rate-variability biofeedback 54
herbal remedies 54
high-risk asthma
care of high-risk patient 70
identifying patients with 70
Home Medicines Review 80, 81
home oxygen therapy 92
homeopathy 54
house dust mites 58, 59, 71, 118, 120
hydrocortisone 39
hydrolysed milk formulae 56, 118
hyperosmolar aerosols 95
hypnotherapy 54
hypoxaemia, chronic 92

ICS dose equivalents 10, 15, 18, 25, 27, 67
ICS therapy 27–9
adjusting maintenance therapy 18–19
during pregnancy 101
effect of cigarette smoking on efficacy 84
for exacerbations 49, 50
for exercise-induced asthma 95
for stable asthma 37
initiation, in adults 18
review regularly 18
role of LABAs in add-on therapy 19, 32
safety 28
starting dose 18
usage 27–8
use in adults 15, 17, 28, 49
use in children 16, 22–3, 28, 50
ICS-LABA combination therapy
adjusting the regimen 19
dosing considerations for exacerbations 49
fixed-dose 21–2, 32
for exercise-induced asthma 96
if response is inadequate 21
use in adults 16, 17, 19
use in children 23
see also combination medications
IgE antibodies, testing for 59, 100
IgE-mediated food allergy 55
IgE-mediated occupational asthma 98
immunomodulatory therapy 61–3
anti-IgE monoclonal antibody therapy 31, 63
conventional (subcutaneous) specific immunotherapy (SIT) in asthma 62–3
objectives 61
other strategies 63
sublingual immunotherapy (SLIT) in asthma 63
immunotherapy
allergic rhinoconjunctivitis in children 120
for allergic rhinitis 61
specific, for asthma 62–3
sublingual, in asthma 63
Indian Ayurvedic medicines 52
infant feed formulas 118
infant feeding, and asthma risk 56, 103, 117–18
infants
asthma prevention 119
dietary allergen avoidance 118
multifaceted interventions to prevent asthma 119
infections, as trigger for asthma exacerbations 72
inflammatory process
effect of 4
potential triggers 4
influenza vaccination 72–3, 110
infrequent intermittent asthma in children 12–13, 14
inhalant allergens as triggers for asthma 58–9
inhalation methods 35
inhaled bronchodilators, for COPD 92
inhaled corticosteroids see ICS therapy
inhaled nasal corticosteroids (INCS), for allergic rhinitis 60
inhaler devices, training in use of 36, 74
inhaler technique 35, 36
reassessment 77
Intal Forte 30

intermittent asthma (adults)
management 17
treated patient 10
untreated patient 8
intermittent asthma (children) 12
frequent 13
infrequent 12–13
International Olympic Committee 97
International Primary Care Respiratory Group (IPCRG) 66
intranasal decongestants, for allergic rhinitis 60–1
Ipratropium bromide 26
dosage 26
for allergic rhinitis 61
nebulised 40
iridology 54
ketotifen 120
kinesiology 54, 60
LABA-ICS combination therapy see ICS-LABA combination therapy
LABAs 4, 26, 32
daily treatment requirements 10
differences in onset of therapeutic action 16, 19
effect on SABA efficacy 21
for exercise-induced asthma 96
principles of drug treatment in adults 15
use in children 16
labour and delivery 102
lactation
dietary allergen avoidance during 118
medications during 103
Lactobacillus acidophilus 53
leukotriene receptor antagonists see LTRAs
life-threatening asthma episode in children, managing 45, 46
lipopolysaccharides 119
long-acting beta2 agonists see LABAs
long-acting inhaled bronchodilators, for COPD 92
lower socio-economic status patients 81
LTRAs 15, 27, 29–30
for allergic rhinitis 60
for exacerbations in children 50
for exercise-induced asthma 96
indications 29–30
use in adults 17, 30
use in children 23, 30, 50
lung function, and CAM 52, 53, 54
lung function tests
forced expiratory volume in one second (FEV1) 7, 93, 94, 121, 122, 123, 125
in adults and adolescents 68
in children 69
in exercise-induced asthma 94–5
in occupational asthma 100
peak expiratory flow 6, 7–8, 11, 48, 68, 69, 100, 124, 126
spirometry 6–7, 11, 48, 68, 69
lycopene 52

Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
magnesium sulphate 40
magnesium supplements 53, 56
maintenance-and-reliever regimen 22
maintenance regimen, standard 22
maintenance therapy, adjusting 18–19
mannitol inhalation 95
manual therapies 53
MDI (Alvesco) 29
MDI (Flixotide) 29
MDI (Intal Forte) 30
MDI (ipratropium bromide) 26
MDI (Qvar) 29
MDI (salbutamol) 26
MDI (Seretide) 33
MDI (Serevent) 32
MDI (Tilade) 31
MDIs 35
use with spacers 36, 37, 39, 50
medication-induced asthma reactions 35
medications
antibiotics 34
antihistamines 34
sedatives 34
that exacerbate asthma 34–5, 71, 76–7
see also asthma medications
medicinal therapies 53–4
mental illness
and adherence to treatment 115
and asthma 114–15
and children with asthma 115
and smoking 115
metabisulfite 72
metered-dose inhaler see MDI
mild acute asthma episode in children, managing 43–4, 45, 46
mild persistent asthma
in children 14
management 20
untreated patient 8–9
milk 55
mineral supplements 52, 53
moderate acute asthma in children, managing 43, 45, 46
moderate persistent asthma
in children 14
management 20
untreated patient 9
monosodium glutamate (MSG) 55, 72
montelukast sodium 29, 30
dosage 30
moulds 58
mucus hypersecretion 34
nebuliser solutions
ipratropium bromide 26
SABAs 26
nedocromil sodium 31
administration 31
dosage 31
for exercise-induced asthma 96
indications 31
nicotine replacement therapy 85
nitrogen dioxide 72
non-immunological occupational asthma 98
NSAIDs 35, 70–71
Nuelin 26
obese patients with asthma, weight loss in 56
obesity, and gastro-oesophageal reflux disease 114
obstructive sleep apnoea 112–13
aetiology 112–13
and gastro-oesophageal reflux 113–14
and obesity 114
clinical implications in asthma management 113
concurrence of snoring, OSA, wheeze and asthma 113
in adults 112
in children 112
treatment 113
occupational asthma 98–100, 120
definition and mechanism 98
diagnosis 99–100
PEF 100
specific immunoglobulin testing 100
incidence 99
information resources 100
management 100
prevention 99
prognosis 100
risk factors 99
ocular anti-allergy preparations 61
omalizumab 31, 63
omega-3 fatty acids 53, 56, 63, 118
ongoing care 64–5
assess asthma control regularly 65–70
identify and avoid trigger factors 70–2
preventing exacerbations 72–3
provide asthma self-management education 73–6
troubleshooting 76–8
ongoing review 70
opportunistic asthma education 75
oral corticosteroids 32, 39
for allergic rhinitis 61
for COPD 92
for severe exacerbations
in adults 48, 49
in children 50
oral decongestants, for allergic rhinitis 61
oral provocation and neutralisation 54
orange inhaler 27
OSA see obstructive sleep apnoea
osteopathy 53
Oxis 32

Panafcort 32
panafcotrernelone 32
paranasal sinus inflammation 57
parenteral corticosteroids 32
passive smoking see environmental tobacco smoke
patient education see self-management education
peak expiratory flow see PEF
peanuts 55
PEF 6, 7–8
diagnosis of asthma 7–8
female children (<18 years) 126
in COPD 91
in occupational asthma 100
male children (<20 years) 124
monitoring in detecting exacerbations 48
use in adults and adolescents 68
use in children 11, 69
persistent asthma
in adults, management 17
in children 13
pet allergens 58, 119
pharmacists, community
Home Medicines Review 80
ongoing monitoring and advice 80
role of 80–1
Team Care Arrangements 81
physical training 53
pilocarpine 71
pneumococcal vaccination 73, 84, 110–11
pneumothorax 40
pollens 58, 59
portable peak flow meters 69
post-partum phase, and asthma 102–3
practice nurses
role of 79
specialised training for effective asthma care 79–80
practice organisation for effective asthma management 78–81
asthma clinics 79
considerations for organisation of asthma review 79
demographic considerations 81
role of community pharmacist 80–1
role of practice nurses 79–80
predictable bronchoconstriction 35
prednisolone 32, 39
prednisone 32
pregnancy and asthma 101–3
antenatal care 102
asthma exacerbations during 102
avoidance of smoking 102, 103
before pregnancy 101
delivery 102
dietary allergen avoidance 118
interventions to prevent asthma 119
medications during pregnancy and lactation 103
post-partum phase 102–3
prescribing devices 35
preparers 4
adherence to regimen 77–8
anti-immunoglobulin therapy 31–2
cromones 27, 30–1
ICS 27–9
LTRAs 27, 29–30
oral or parenteral corticosteroids 32
principles of treatment
in adults 15
in children 16
use in children 24
preventing exacerbations 72–3
prevention of asthma 116–20
primary prevention 117–19
allergen avoidance and reduced asthma risk 118–19
environmental factors 119
exposure to environmental tobacco smoke and asthma risk 117
infant feed formulas 118
infant feeding and asthma risk 117–18
probiotics 63, 118
psionic medicine 54
psychological therapies 54
psychosomatic illness 114
Pulmicort 27, 29
pulmonary rehabilitation, in COPD 92
pulse tests 54, 60
pyridostigmine 71
qigong 53
quit smoking
advice and counselling 84
clinical interventions 84–6
drug therapy 85
general practice guidelines 86
other strategies 85–6
pneumococcal vaccination 84
public policy initiatives 87
Qvar 27, 28–9
radioallergosorobent tests see RAST testing
radionics 54
RAST testing 56, 59–60
recurrent non-specific cough, in children 12
referral
to allergy specialist 61, 71
to ENT specialist 61
to respiratory paediatrician, children and adolescents 78
to respiratory specialist, adult patients 78
reflexology 54
relaxation therapies 54
relevers 4, 25–7
adherence to regimen 77–8
in adults 15, 17
in children 16
ipratropium bromide 26
LABAs 4, 10, 16, 19, 21, 26, 32, 96
SABAs 10, 16, 17, 25–6, 39, 48–9, 50, 96
Copyright National Asthma Council Australia 2006. Non-profit reproduction for patient counselling or educational purposes only is permitted.
Index

theophylline 26–7
respiratory function tables
  mean predicted normal values for healthy adults 121–2
  mean predicted normal values for healthy children and adolescents 123–6
respiratory paediatrician, referral to 78
respiratory specialists, referral to 78
Respules (budesonide) 29
rhinitis medicamentosa 61
royal jelly 52, 71
rural and remote regions 81
rust-coloured inhaler 27
ryegrass 59

SABAs 25–6
  as reliever therapy
    in adults 17
    in children 16
  as short-term reliever for intermittent asthma 17
daily treatment requirements 10
delivery 25
for COPD 92
for exercise-induced asthma 96
for managing exacerbations 48–9
indications 25
IV aminophylline as alternative to 40
LABA use effect on efficacy 21
methods of action and use 25
principles of drug treatment 15, 16
use in spirometry 6
  via MDI plus spacer
    for acute asthma 39
    for exacerbations 48, 50
salbutamol 25
dosage and use 26
salbutamol dose equivalents 45
salicylate derivatives 55
salmeterol 32
  during pregnancy 103
  for COPD 92
  see also fluticasone plus salmeterol
secondary prevention 120
  in adults 120
  in children 1120
sedatives 34
selenium supplements 53, 56
self-management, what is it? 74
self-management education 4, 73–6
  as part of comprehensive care program 75–6
  asthma action plans 74–5, 127–8
checklist 129
for adolescents 75
health professionals role 73
in elderly patients 109
information resources 76
opportunistic asthma education 75
purpose 73
structured programs 74
training in correct use of inhaler devices 36, 74
self-management proficiency review 77
assess attitudes 77
check adherence 77–8
reassess inhaler technique 77
Seretide 29, 32
dosage for exacerbations 49
dose adjustment 22
onset of therapeutic action 16, 19, 33
Serevent 32
severe acute asthma in children, managing 43, 45, 46
severe persistent asthma
  in children 14
  management 20
  treated patient 10
  untreated patient 9
severity assessment see assessment of severity
shellfish 55
'short wind' 6
short-acting beta2 agonists see SABAs
Singular 29, 30
SIT see specific immunotherapy
Six Step Asthma Management Plan 1, 131
skin prick tests 56, 59, 100
SLIT see sublingual immunotherapy in asthma
smog 72
smoke-free zones, making car and home 83
smokers, pneumococcal vaccination 84
smoking
  and asthma 82–7, 120
  and mental illness 115
  as risk factor for COPD 89
clinical interventions to help patients quit smoking 84–6
effect of active smoking in people with asthma 83
mechanisms for effects on asthma 83–4
public policy 86–7
see also environmental tobacco smoke
smoking cessation
  before pregnancy 101, 103
GP time expenditure 85
guidelines for Australian general practice 86
in COPD management 92
promotion of 87
strategies 84–6
smoking rates
  among people with asthma 83
  in Australia 82–3
smoking uptake, influences on 86
sodium cromoglycate 30
administration 30
dosage 30
  for exercise-induced asthma 96
indications 30
sodium diet, low 56
soy milk formula, in infants 56, 118
spacers
and MDIs 36, 37, 39, 50
use and care of 36
use with SABAs 39, 48, 50
specific immunotherapy in asthma 62–3
adverse effects 62–3
indications for 62
spirometry 6–7, 48, 63
in COPD 91, 107–8
interpreting 7
overlap of asthma and COPD 108
performance method 6–7
purpose of 6
use in adults and adolescents 68
use in children 11, 69
use in elderly 107–8
sputum eosinophilia test 8
stable asthma
beta: agonist delivery for 37
ICS delivery for 37
structured self-management programs 74
Student Asthma Record 132
sublingual immunotherapy in asthma 63
sulphur dioxide 55
swimming 53
Symbicort 15, 33, 34
dose adjustment 22
doctor consideration for exacerbations 49
onset of therapeutic action 16, 19, 33
use as reliever 17
Symbicort Turbuhaler 34
symptom controllers 4, 32
LABAs 32
systemic corticosteroids 39
tai chi 53
tartrazine 55
Team Care Arrangements 81
terbutaline 25
doctor and use 26
Terry White Chemists’ Ipratropium 26
tertiary prevention 120
theophylline 26–7
concentration monitoring 27
doctor and use 27
indications 27
3+ Visit Plan 79, 131
Tilade 31
tiotropium 92
traditional Chinese medicine 52, 54
training in correct use of inhaler devices 36, 74
treated asthma patients, classification of asthma severity 10
tree nuts 55
tree pollen 58
trigger factors 4, 71–2, 118–19
air environment 72
air pollutants 72
allergens 58–9, 71, 77
foods 72, 118
gastro-oesophageal reflux 72
medications 71, 76
reassessment 76–7
troubleshooting 76–8
checklist 76
reassess triggers 76–7
review self-management proficiency 77–8
review the history 77
when to refer to a specialist? 78
Turbuhaler, as drug delivery device 35
Turbuhaler (budesonide) 29
Turbuhaler (Oxis) 32
Turbuhaler (Symbicort) 34
Turbuhaler (terbutaline) 26
unpredictable medication-induced asthma exacerbations 35
untreated asthma classification 8–10
intermittent asthma 9
mild persistent asthma 8–9
moderate persistent asthma 9
severe persistent asthma 9
vaccinations 72–3, 84, 110–11
vega matrix regeneration therapy 54
Vega tests 54, 60
Ventolin 25
viral bronchiolitis 23
vitamin C 53, 56
vitamin E 53, 56
vitamin supplements 52
volatile organic compounds 72
weed pollen 58
weight loss in the obese patient 56
wheezing
concurrence of snoring, OSA, wheeze and asthma 113
in adults 5, 6
in children 11, 23, 69
willow tree bark extracts 52
Written Asthma Action Plan 131
Xolair 31
yoga 53
The National Asthma Council Australia has received generous support from the following bodies for the publication of this document.
The Asthma Management Handbook aims to be both comprehensive and user-friendly. While written primarily for general practitioners and community pharmacists, the Handbook emphasises a team approach to asthma care. It contains:

- updated diagnostic, management and prescribing guidelines
- expanded material on asthma and allergy, exercise-induced asthma, occupational asthma, asthma in pregnancy and in older people, and comorbidities
- more detail on diet and complementary medicine
- new chapters on smoking cessation and asthma prevention
- practical advice on providing structured asthma care in the primary care setting.

Visit the National Asthma Council Australia website for full references for this document, further information and resources, and links to other asthma and respiratory sites:

www.nationalasthma.org.au

ISSN 1325-4405