Fiji Respiratory Guidelines

2nd Edition, 2022

Ministry of Health and Medical Services

Government of Fiji

Therapeutic Guidelines

World Health Organization

Ministry of Health and Medical Services, Government of Fiji

These *Fiji Respiratory Guidelines* have been endorsed by the National Medicines and Therapeutics Committee, Ministry of Health and Medical Services, Government of Fiji.

Medicines recommended in these guidelines that are not currently included on the Fiji *Essential Medicines List* (EML) are denoted by [non-EML]. The medicines included on the EML may change during the lifetime of these guidelines—refer to the most up-to-date EML for more information.

For feedback on these guidelines, please email shrish.acharya@health.gov.fj.

These guidelines have been adapted from *Therapeutic Guidelines: Respiratory* with permission. Therapeutic Guidelines is an independent not-for-profit organisation which develops and publishes evidence-based, practical treatment advice to assist practitioners with decision making at the point-of-care. For more information or to discuss permission to adapt Therapeutic Guidelines content, email partnershipprogram@tg.org.au.

Contents

Tables

Boxes

Figures

Images

Foreword

The first edition of the Fiji Respiratory Guidelines was published in 2008 which has been reviewed and revised by the Respiratory Guideline Committee to produce this second edition. The Committee has based this second edition of the Fiji Respiratory Guidelines on the Australian *Therapeutic Guidelines: Respiratory*, version 5, 2015 and updated with the publication of *Therapeutic Guidelines: Respiratory*, version 6, 2020.

This new edition provides the reader with a holistic approach to the management of respiratory diseases. Not only does the Guideline incorporate pharmacological therapies but also includes non-pharmacological aspects of treatment of respiratory diseases to provide users of this guideline with comprehensive treatment options for their patients.

Some of the features of this edition includes:

- Broader coverage of respiratory diseases
- Chest X-ray images for easy reference and recognition
- Advice on when to refer the patient to the next level of health care.
- Information on asthma action plans including templates

The medicines stated in this Guideline are mostly those that are available on the Fiji Essential Medicines List (EML), however, there are some medications that are not available on the EML and these are clearly stated. Generally, these have been included as they are widely available in the private health sector in Fiji. All recommended therapies are either reference based or universally accepted standards.

It is hoped that this Guideline will be a reference for all healthcare workers, both in the public and private sector, to care for patients suffering from respiratory diseases. The Respiratory Guideline Committee welcomes any comments and suggestions, which will help in the improvement of the development of future standard treatment guidelines. Please forward your feedback to <shrish.acharya@health.gov.fj>.

I believe that these guidelines will promote quality care for patients in Fiji and trust that they will be widely used by all prescribers.

Dr James Fong

Permanent Secretary, Ministry of Health and Medical Services

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This second edition of the Fiji Respiratory Guideline has been based on the Australian *Therapeutic Guidelines: Respiratory*, version 5, 2015 and updated with the publication of *Therapeutic Guidelines: Respiratory*, version 6, 2020. The Fiji Respiratory Guideline Committee gratefully acknowledges the permission to use the Guidelines in 2016 from Sue Phillips, (then) Chief Executive Officer of Therapeutic Guidelines Limited, without levy of any fee, and expresses appreciation to Professor Robert Moulds, Chairman of the Respiratory Expert Group and Medical Advisor for the Therapeutic Guidelines Limited, for the first draft of this edition. The Fiji Respiratory Guideline Committee updated this edition of the Respiratory Guideline and acknowledges contributions and comments from:

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Respiratory symptoms occurring in children

Extensive information on the assessment of respiratory symptoms in children is contained in the *Pocket book of hospital care for children: guidelines for the management of common childhood illnesses* ('WHO Blue Book'). Below are brief summaries of important respiratory symptoms in children.

Wheeze

Wheeze is produced by turbulent airflow in the lower airways. It is most commonly heard in expiration and can be acute or chronic. Parents should be asked specifically about the presence of a high-pitched whistle on expiration. In the first year of life, wheeze is commonly due to conditions other than asthma; hence, wheezy infants should not automatically be treated for asthma.

Asthma and bronchiolitis are the most common causes of acute expiratory wheeze in children. There are several other conditions that can cause chronic wheeze. For example, a thriving infant with expiratory wheeze from the first few weeks of life is likely to be due to mild tracheomalacia. These children are often referred to as 'fat happy wheezers.' Alternatively, wheeze that occurs for the first time at 3 to 4 months of age, worsens with time, and is associated with increased work of breathing and choking or gagging on feeds, should trigger referral to a specialist paediatrician at a divisional hospital.

Referral is indicated for most infants with chronic persistent wheeze.

Cough

As in adults, the most common cause of cough in children is acute viral respiratory tract infection. The cough can be wet or dry, and symptoms settle spontaneously in 7 to 10 days. Oral antibiotic treatment is not required.

A daily wet cough that persists for longer than 3 weeks is suggestive of 'persistent bacterial bronchitis' and referral for assessment is recommended. A chronic wet cough can also be a sign of suppurative lung disease such as bronchiectasis.

Cough can occur in children with asthma. However, cough alone is a poor marker of asthma, which should not be diagnosed in the absence of other symptoms of airway obstruction (eg recurrent wheeze, tachypnoea and dyspnoea).

See the separate topic "Cough" for further information on cough in children, including alarm symptoms and findings.

Stridor

Stridor is a respiratory noise produced by turbulent airflow through the upper airways. It is most commonly heard in inspiration and can be acute or chronic.

The most common causes of acute stridor in children include viral laryngotracheobronchitis (croup), acute tonsillitis (with or without peritonsillar abscess) and epiglottitis. This is rare as *Haemophilus influenzae* type B vaccine is given routinely in Fiji. Foreign body inhalation should be suspected in children who present with acute stridor after a choking episode.

Some children present with a chronic 'cog-wheel' high-pitched inspiratory stridor that has been present from birth or the first few days or weeks of life. The most common cause is laryngomalacia. The stridor resolves spontaneously at 1 to 2 years of age. Alternatively, inspiratory stridor that develops for the first time at 6 to 8 weeks of life, worsens and becomes biphasic (present in both inspiration and expiration) may be due to a subglottic haemangioma.

All infants and children with stridor need to be discussed with specialist.

Respiratory symptoms occurring in adults

Wheeze

Wheeze in adults is produced by turbulent airflow in the lower airways, is most commonly heard in expiration, and can be acute or chronic. If intermittent wheeze is suspected, patients should be asked specifically about the presence of a high-pitched whistle on expiration.

Although asthma is the most common cause of both acute and chronic expiratory wheeze in adults, other common causes are COPD, acute bronchitis, bronchiecstasis, bronchostenotic lesions, heart failure and hypersensitivity reactions. It should not be assumed that wheeze is necessarily caused by asthma.

Stridor

Stridor in adults is a harsh respiratory noise produced by turbulent airflow through the upper airways – usually the trachea or main bronchi – and is usually heard in inspiration.

It is a serious symptom which is usually caused by a significant lesion causing partial obstruction of the trachea or a major bronchus such as a bronchial carcinoma.

All patients with stridor require urgent referral to a divisional hospital for further assessment.

Cough

The most common cause of cough in an adult is an acute viral respiratory tract infection. The cough can be wet or dry, and symptoms usually settle spontaneously in 7 to 10 days. Persistent cough is difficult to diagnose and treat and requires referral to a divisional hospital. Oral antibiotic treatment is **not** required.

Refer to the separate topic “Cough” (page 111) for a more detailed discussion.

Shortness of breath

Shortness of breath, also termed dyspnoea, is probably the most common and important respiratory symptom in adults.

Depending on the cause, it can be of acute onset, progressive in nature, or episodic. In most individuals, a detailed history and physical examination with appropriate investigations (which include baseline blood tests, a chest X-ray and an ECG) will reveal the underlying cause of dyspnoea. A few patients may require further intensive investigations and they warrant referral to a divisional hospital.

There are many causes of shortness of breath in an adult; some are physiological, such as aging and obesity, while others are pathological conditions that fall under the following categories:

- respiratory diseases
- cardiac diseases
- anaemia
- thyrotoxicosis
- metabolic acidosis
- psychogenic – to be considered when organic causes have been ruled out

Heart failure and chronic obstructive pulmonary disease (COPD) are common causes of persisting shortness of breath in adults. Multiple causes can co-exist, especially in older adults. Individuals not responding to treatment should be discussed with a specialist and may require referral for further assessment.

Asthma in children (up to 15 years)

General information

This topic addresses the management of asthma in children from 1 to 15 years; for adolescents, see 'Asthma in adults and adolescents,' page 32.

- asthma is a chronic inflammatory disease of the airways characterised by reversible airways obstruction and bronchospasm.
- exacerbations in children are often precipitated by viral infection
- in children less than 12 months of age presenting with wheeze, consider the diagnosis of bronchiolitis.
- early recognition and acute management of severe or life-threatening disease is of vital importance.

Most children diagnosed with asthma in Fiji have intermittent symptoms triggered by viral respiratory tract infection. Treatment with inhaled salbutamol on an 'as required' basis is usually sufficient to control symptoms, which improve with age. In the minority of children with asthma who require preventive treatment with an inhaled corticosteroid, care should be taken to use the lowest dose that controls asthma symptoms. Children with recurrent cough alone are often misdiagnosed with asthma; see below for further information.

Diagnosis

There is no single reliable test ('gold standard') and there are no standardised diagnostic criteria for asthma.

The diagnosis of asthma is based on:

- history
- physical examination
- considering other diagnoses
- clinical response to a treatment trial with an inhaled short-acting $beta_2$ agonist reliever or preventer

Patient history should include:

- current symptoms (wheeze, cough, shortness of breath, chest discomfort or tightness)
- pattern of symptoms (frequency, time of day or night)
- severity of symptoms (impact on work, school or lifestyle)
- allergies (eg atopic dermatitis, allergic rhinitis)

- aggravating or precipitating factors (eg exercise, viral infections)
- smoking history (including exposure to second-hand smoke in the home) and exposure to biomass smoke (eg indoor fires for heating or cooking)
- relieving factors (including medication trials)
- presence of sinonasal disease
- family history of allergies or asthma.

The physical examination should include:

- chest auscultation
- height and weight
- inspection for chest deformity
- assessment of respiratory rate and work of breathing.

If atopy is suspected, inspect the upper respiratory tract for signs of allergic rhinitis (eg inflammation in the nasal passages) and the skin for signs of atopic dermatitis.

A chest X-ray is not necessary for diagnosis of asthma—it can be considered for unusual symptoms, or as required for suspected alternative diagnoses (eg lung cancer, pneumonia).

An episode of acute asthma can present as a wheeze precipitated by triggers such as tobacco smoke, pets (eg cats, dogs), dust, and other allergens (see Table 7 for list of possible triggers). This is often associated with atopic diseases including allergic rhinitis or atopic dermatitis in children. Table 1 lists features that increase and decrease the likelihood of asthma. Although the probability of asthma does not necessarily change initial management, it can be a useful part of the discussion with parents and carers.

Age 0 to 12 months

Urgently refer infants with sudden-onset wheeze if foreign body aspiration or anaphylaxis is suspected. Acute wheeze in an infant younger than 12 months is most commonly a symptom of acute bronchiolitis. Asthma cannot be diagnosed in this age group, and infants should not be treated with asthma medication.

Age 1 to 5 years

Although many individuals later diagnosed with asthma first show respiratory symptoms by the age of 5 years, it is difficult to make the diagnosis of asthma with a high degree of certainty in children aged 1 to 5 years, because:

- episodic respiratory symptoms such as wheezing and cough are very common in children, particularly in children under 3 years

- objective lung function testing by spirometry is usually not feasible in this age group
- a high proportion of children who respond to bronchodilator treatment do not go on to have asthma in later childhood (eg by primary school age).

Table 1: Clinical features that increase and decrease the probability of children 1 to 5 years with wheeze having asthma in later childhood and adulthood

CLINICAL FEATURES THAT INCREASE THE PROBABILITY OF ASTHMA
• more than one of the following symptoms: wheeze, breathlessness, chest tightness or discomfort, cough – particularly if symptoms: – are worse at night – occur in response to active play, laughing, allergen exposure or cold air – are recurrent • wheeze occurring when the child does not have a cold • history of atopic disorder (eg allergic rhinitis, atopic dermatitis) • family history of asthma or atopic disorder • widespread wheeze heard on auscultation of the chest • improvement in symptoms in response to trial of asthma therapy • otherwise unexplained peripheral blood eosinophilia • presence of conditions associated with asthma (eg bronchopulmonary dysplasia, obstructive sleep apnoea, recurrent bronchiolitis)
CLINICAL FEATURES THAT LOWER THE PROBABILITY OF ASTHMA
• chronic productive cough in the absence of wheeze or breathlessness • repeatedly normal auscultation of chest when symptomatic • voice disturbance or throat tightness • prominent dizziness, light-headedness, peripheral tingling • no response to a trial of asthma therapy • clinical features supporting an alternative diagnosis

Age 6 to 15 years

The first step in assessing a child older than 6 years, is to take a detailed history and perform a physical examination to identify the pattern of symptoms and exclude other causes. The predictive value of a single sign or symptom is poor, but combinations of signs and symptoms can provide a clearer clinical picture to support a diagnosis of asthma.

Table 2: Clinical features that increase and decrease the probability of asthma in children 6 years and older

INITIAL CLINICAL ASSESSMENT
Focus the initial assessment in children suspected of having asthma on: • presence of key features in history and examination • careful consideration of alternative diagnoses (see table below).
CLINICAL FEATURES THAT INCREASE THE PROBABILITY OF ASTHMA
• more than one of the following symptoms: wheeze, breathlessness, chest tightness or discomfort, cough—particularly if symptoms: – are worse at night and in the early morning – occur in response to exercise, allergen exposure or cold air – occur after taking aspirin or beta blockers – are recurrent • history of atopic disorder (eg allergic rhinitis, atopic dermatitis) • family history of asthma or atopic disorder • widespread wheeze heard on auscultation of the chest • improvement in symptoms or lung function in response to standard asthma therapy • otherwise unexplained low FEV_1 or PEF (historical or serial readings) • otherwise unexplained peripheral blood eosinophilia • in children, presence of conditions associated with asthma (eg bronchopulmonary dysplasia, obstructive sleep apnoea, recurrent bronchiolitis)

cont...

CLINICAL FEATURES THAT LOWER THE PROBABILITY OF ASTHMA

- chronic productive cough in the absence of wheeze or breathlessness
- normal FEV_1 when symptomatic [Note 1]
- repeatedly normal auscultation of chest when symptomatic
- voice disturbance or throat tightness
- symptoms that worsen with talking or laughing
- prominent dizziness, light-headedness, peripheral tingling
- symptoms that only occur with viral respiratory infections, with few or no symptoms in between
- no response to a trial of asthma therapy
- clinical features supporting an alternative diagnosis

Record the basis on which a diagnosis of asthma is suspected.

FEV_1 = forced expiratory volume in 1 second; PEF = peak expiratory flow

Note 1: Normal spirometry when the patient is not symptomatic does not exclude the diagnosis of asthma; ideally, repeat spirometry when the patient is symptomatic. If spirometry is normal when the patient is symptomatic, consider an alternative diagnosis. Repeated measurements of lung function are often more informative than a single assessment.

Table 3: Alternative diagnoses other than asthma to consider in children according to predominant symptoms

Symptom	Possible alternative diagnosis
dry cough	postinfective cough (respiratory viruses, *Bordetella pertussis* or *Mycoplasma pneumoniae*)
	habit cough, particularly if it resolves during sleep
wheeze	virus-associated wheeze
	in young children: • transient infant wheeze • tracheobronchomalacia • airway lesion • inhaled foreign body (unilateral wheeze) • cardiac left-to-right shunt
difficulty breathing	chronic lung disease of prematurity (bronchopulmonary dysplasia)
	cardiac left-to-right shunt
	upper airway dysfunction (vocal cord dysfunction)

cont...

chest tightness	Anxiety
wet cough (with sputum production in older children)	persistent bacterial bronchitis chronic suppurative lung disease (including bronchiectasis) cystic fibrosis

Maintenance management of asthma in children

Overview

Most children with asthma will experience improvement in symptoms with age.

The aim of management of asthma in children is to maintain a normal quality of life, free of asthma symptoms and without adverse effects of asthma treatment. Maintenance management relies on a continuous cycle of reviewing response and adjusting therapy, aiming to establish the minimum drug regimen that achieves good control.

Specific questions should be asked about sleep disturbance (due to asthma), early morning symptoms, exercise induced cough or wheeze, and frequency of bronchodilator use.

Spacers

- A spacer device should be used for children of all ages whenever they use a metered dose inhaler (puffer).
- Small volume spacers should be fitted with a well-sealing face mask for infants who cannot reliably seal their lips around the mouthpiece.
- Large volume spacers should not be used for children under 6 years - they can be used above this age but are more cumbersome and less convenient than the smaller ones.

Small volume spacers are recommended for children of all ages.

Control-based management

The aim of asthma management in children is to achieve good symptom control and prevent flare-ups (exacerbations) using the lowest effective preventer dose. Asthma symptom control is assessed according to the frequency of asthma symptoms over the previous 4 weeks. This allows definition of control as good, partial or poor; see table below.

Table 4: Levels of recent asthma symptom control in children [Note 1]

Good control	Partial control	Poor control
All of the following features: • daytime symptoms (eg wheezing or breathing problems) on **2 or fewer days per week**, lasting only a few minutes and rapidly relieved by short-acting bronchodilator • no limitation of activities; child is fully active, runs and plays without symptoms • no symptoms during night or on waking, including no coughing during sleep • need for reliever on **2 or fewer days per week** [Note 2]	Any of the following features: • daytime symptoms (eg wheezing or breathing problems) on **more than 2 days per week**, lasting only a few minutes and rapidly relieved by short-acting bronchodilator • any limitation of activities; wheeze or breathlessness during exercise, vigorous play or laughter • any symptoms during night or on waking (eg waking with symptoms of wheezing or breathing problems) • need for reliever **on more than 2 days per week** [Note 2]	Either of the following features: • daytime symptoms (eg wheezing or breathing problems) on more than 2 days per week, lasting from minutes to hours or recurring, and partially or fully relieved by short-acting bronchodilator • three or more features of partial control within the same week

Note 1: Recent asthma symptom control is based on symptoms over the previous 4 weeks irrespective of the current treatment regimen.

Note 2: Not including short-acting beta$_2$ agonist taken prophylactically before exercise; record this separately and take into account when assessing management.

Source: National Asthma Council Australia. Australian Asthma Handbook, Version 1.0. Melbourne: National Asthma Council Australia; 2014. [http://www.asthmahandbook.org.au/]

Drug treatment

Stepwise approach to treatment in children

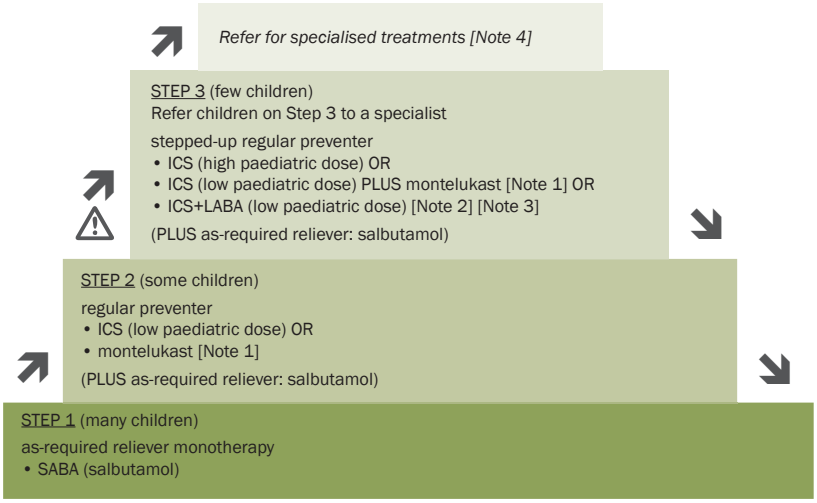
Overview

A stepwise approach to treating asthma in children is shown below. It applies to children with intermittent asthma and persistent asthma.

Asthma severity is defined by the intensity of treatment required to achieve good asthma symptom control. It is a retrospective label that is applied after a child has been using preventer treatment for at least 3 to 6 months. Children are considered to have mild asthma when good symptom control is achieved using optimised treatment at Step 1 or Step 2 (see Figure 1). Children are considered to have moderate or severe persistent asthma when symptoms do not respond to optimised treatment at Step 3 (see Figure 1); referral to a specialist paediatrician is recommended.

For detail of drugs used to treat asthma in children, see the next section: 'Drug doses and administration in children.'

Figure 1: Stepped approach to adjusting asthma medication in children 15 years and younger.



⚠	Before considering stepping up treatment, review adherence and inhaler technique, check equipment (eg inhaler, spacer, mask) for breakage or blockage, assess for an alternative diagnosis or a comorbidity (eg rhinitis), and ensure exposure to triggers is minimised.
↗	Consider stepping up if good control is not achieved despite good adherence and correct inhaler technique; see Assessment of asthma control in children for more information.
↘	*If asthma has been stable and well controlled for at least 3 months, consider stepping down therapy; see Stepping down asthma therapy in children for more information.*

cont...

ICS = inhaled corticosteroid; LABA = long-acting $beta_2$ agonist; SABA = short-acting $beta_2$ agonist

Note 1: Montelukast is less effective than ICS and has been associated with neuropsychiatric adverse effects, including nightmares, sleep disturbance, agitation, depression and, rarely, suicidal thinking and behaviour. Counsel parents and carers about these effects. They usually occur in the first 2 weeks of treatment. Stop treatment if these effects occur—the effects usually subside within a few days of stopping treatment. See the Australian Therapeutic Goods Administration (TGA) website for more information: www.tga.gov.au/alert/montelukast

Note 2: Always give ICS+LABA therapy as a fixed-dose combination inhaler to avoid the possibility of patients taking a LABA without an ICS; LABA monotherapy increases the risk of exacerbations and asthma-related death.

Note 3: There is no evidence to support the use of LABAs in children younger than 5 years; use in this age group is not recommended.

Note 4: Children who remain uncontrolled despite Step 3 therapy (with good adherence and inhaler technique, and no likely alternative diagnoses) are considered to have severe asthma. These children require referral to a specialist (paediatrician) for investigation and management.

Additional references:

Australian Asthma Handbook © 2020 National Asthma Council Australia. Accessed January 2022.

Paul V, Bagga A, editor. GHAI Essential Pediatrics. 8th ed. New Delhi: CBS Publishers and Distributors Pvt Ltd; 2013: 387.

Table 5: Stepwise treatment of asthma in children 15 years and younger

[Note 1]

Step 1
Intermittent
salbutamol 100 micrograms per puff (actuation) as required
child 1 to 5 years: 2 to 6 inhalations via MDI with spacer (and face mask if required)
child 6 to 15 years: 2 to 12 inhalations via MDI with spacer
If symptoms remain consistent with partial control, eg daytime symptoms on more than 2 days/week, move to Step 2.

cont...

Step 2

Mild persistent

salbutamol 100 micrograms per puff (actuation) as required

child 1 to 5 years: 2 to 6 inhalations via MDI with spacer (and face mask if required)

child 6 to 15 years: 2 to 12 inhalations via MDI with spacer

PLUS **one** of the following:

inhaled corticosteroid (low dose)

eg beclomethasone 100 micrograms by inhalation via MDI with spacer ONCE or TWICE daily

OR

montelukast

Child 1 to < 6 years: 4 mg daily

Child 6 to 15 years: 5 mg daily

Step 3

Moderate to severe persistent

Any child with moderate persistent asthma should be referred to a specialist for management

salbutamol 100 micrograms per puff (actuation) as required

child 1 to 5 years: 2 to 6 inhalations via MDI with spacer (and face mask if required)

child 6 to 15 years: 2 to 12 inhalations via MDI with spacer

PLUS one of the following:

inhaled corticosteroid (**high dose**)

eg beclomethasone 100 to 200 micrograms (maximum dose 400 micrograms per day in severe cases) by inhalation via MDI with spacer twice daily

OR

inhaled corticosteroid (low dose) PLUS montelukast (refer to doses above)

OR

combination low dose inhaled corticosteroid and long-acting beta-2 agonist (ICS-LABA given as a fixed-dose combination) non-EML

Note 1: See the next section: 'Drug doses and administration in children,' for specific drug and dose recommendations.

Children who remain uncontrolled despite Step 3 therapy (with good adherence and inhaler technique, and no likely alternative diagnoses) are considered to have severe

asthma. These children require referral to a specialist (a paediatrician or paediatric respiratory physician) for investigation and management.
Specialist management of asthma may include the addition of theophylline or tiotropium (non-EML) to standard therapy. Theophylline should only be initiated by a specialist and ideally requires therapeutic drug monitoring.

Drug doses and administration in children

Short-acting beta$_2$ agonists

Prescribe as-required reliever therapy for all children with a diagnosis of asthma. Use:

child 1 to 5 years: 2 to 6 inhalations via MDI with spacer (and face mask if required)

child 6 to 15 years: 2 to 12 inhalations via MDI with spacer.

Educate parents and carers about how to use the inhaler, including advice about using a spacer (recommended for all children using a MDI) and a mask (if required for children using a MDI). See page 197 for links to instructional videos and patient handouts for devices.

A SABA is referred to as a 'reliever'; the term 'rescue medication' may be used in the literature.

Inhaled corticosteroids

Inhaled corticosteroids (ICS) (beclomethasone) are the most effective preventive therapy in children with asthma. They are referred to as 'preventers' and are first-line maintenance treatment.

To minimise the risk of oropharyngeal candidiasis and systemic corticosteroid absorption, children should be advised, or helped, to rinse their mouth with water and spit out straight after using inhaled corticosteroids (ICS). Using a spacer with ICS also reduces oropharyngeal candidiasis and dysphonia.

- before considering stepping up, ensure correct diagnosis, control of trigger factors, correct inhaler technique, compliance to treatment plan
- consider stepping up if good control is not achieved
- when asthma is well controlled and stable for at least 3 months, consider stepping down (reduce dose or stop ICS)
- oral corticosteroids should **not** be prescribed long term for the treatment of asthma – for children with moderate to severe persistent asthma, refer to a specialist for management.

For children requiring preventer treatment in addition to as-needed reliever treatment (ie Step 2 in figure above), use:

a low-dose ICS by inhalation, see below.

Review treatment after 2 to 3 months to determine the level of asthma symptom control. Low and high doses of ICS for children are presented below.

Use the minimum effective dose of ICS to reduce the risk of adverse effects.

Explain to parents and carers that ICS therapy needs to be used every day to be effective and does not relieve acute symptoms.

Educate parents and carers about how to use the inhaler, including advice about using a spacer (recommended for all children using a MDI) and a mask (if required for children using a MDI). See 'Inhalational drug delivery devices,' page 197 for information about using masks and spacers.

Table 6: Corticosteroid-based-inhalers available in Fiji for asthma in children

Drug [Note 1]	**Dosage**	
	Low paediatric dose (Step 2)	**High paediatric dose (Step 3)** [Note 3]
beclometasone	100 to 200 micrograms **per day** given in one or two doses [Note 2]	200 micrograms twice daily
budesonide (including combinations with LABA) Non-EML	100 to 200 micrograms twice daily	300 to 400 micrograms twice daily
fluticasone propionate (including combinations with LABA) Non-EML	50 to 100 micrograms twice daily	125 to 250 micrograms twice daily

LABA = long-acting beta agonist

Note 1: For information about delivery devices, including links to videos and patient handouts, see Inhalational drug delivery devices page 197.

Note 2: A 50 microgram MDI is currently not available on the Fiji EML; the 100 microgram MDI can be used once daily.

Note 3: Doses above the upper limit of the high dose range should not be prescribed without specialist advice.

Montelukast

Montelukast (a leukotriene receptor antagonist) is a tablet used as an alternative to inhaled corticosteroids (ICS). Montelukast is not on the Fiji EML but is available in the private sector. It may be trialled if:

- the child is unable to use inhaled therapy
- the child also has significant allergic rhinitis

- the parents have strong concerns about adverse effects of ICS.

  montelukast (2 to 5 years) 4 mg orally, at night
  or montelukast (6 to 14 years) 5 mg orally, at night.

Montelukast has been associated with neuropsychiatric adverse effects, including nightmares, sleep disturbance, agitation, depression and, rarely, suicidal thinking and behaviour. Counsel parents and carers about these effects. They usually occur in the first 2 weeks of treatment. Stop treatment if these effects occur—the effects usually subside within a few days of stopping treatment. See the Australian Therapeutic Goods Administration (TGA) website for more information: www.tga.gov.au/alert/montelukast.

Theophylline

Theophylline should only be initiated by a specialist and children taking theophylline must reviewed regularly.

Theophylline has a narrow therapeutic range, the dose should be adjusted according to clinical response and plasma concentrations (monitoring of theophylline plasma concentration is currently not available in Fiji). Educate patients or carers about common symptoms of toxicity such as nausea, vomiting, diarrhoea, tremor or palpitations, and to seek medical attention if they experience any of these.

Ideal body weight should be used for dose calculations in obese patients.

Cromones

Cromones (cromoglycate, nedocromil) are rarely used alternative preventer therapies. Cromones are not as effective as ICS and require more frequent dosing and meticulous daily care to prevent clogging of the inhaler device. Nedocromil has not been studied in children 1 to 5 years. There are no cromones listed on the Fiji EML at the time of publication.

Exercise-induced bronchoconstriction

In school-aged children with asthma, exercise is usually one of a number of triggers for bronchoconstriction. Less commonly, it may be the only, or predominant, trigger. The level of physical activity needed to trigger exercise-induced bronchoconstriction depends on the child, and also on ambient environmental factors (symptoms are more likely in cold and dry conditions).

For children with persistent asthma, regular preventer therapy is the most important factor in controlling exercise-induced bronchoconstriction. In children in whom exercise is the only trigger for asthma symptoms, pre-exercise bronchodilator therapy is usually effective. Use:

salbutamol 100 micrograms per puff (actuation), 1 to 2 puffs by inhalation via MDI with spacer, 15 minutes before exercise.

Nondrug interventions

Avoiding triggers for children with asthma is important. Exposures that increase risk of children developing asthma are discussed above.

Respiratory viral infection is the most common trigger in childhood asthma. Exposure to airborne allergens can also trigger asthma symptoms in sensitised children, see Table 7.

There is no good evidence for use of complementary medicines to treat asthma in children. Some complementary medicines (eg royal jelly, echinacea) are known triggers for asthma and anaphylaxis.

Table 7: Management of triggers for flare-ups of existing asthma in children

[Note 1]

Action	Trigger
Always avoid	cigarette smoke
Avoid or minimise if possible	allergens (eg pollen, dust mite)
	airborne or environmental irritants (eg cold or dry air, occupational irritants, pollution, smoke)
	drugs associated with asthma exacerbations (eg NSAIDs for patients with aspirin-exacerbated respiratory disease, beta blockers [Note 2])
	dietary triggers (either temperature related [eg cold drinks] or allergy related [for patients with food allergies]) [Note 3]
Manage	respiratory tract infections
	comorbidities (eg allergic rhinitis, gastro-oesophageal reflux, nasal polyposis, obesity, inducible laryngeal obstruction)
	physiological and psychological changes (extreme emotions, hormonal changes, pregnancy, sexual activity)

NSAID = nonsteroidal anti-inflammatory drug

Note 1: No individual item triggers asthma in all people.

Note 2: If a patient with asthma develops an indication for beta-blocker therapy (eg heart failure, myocardial infarction), start beta-blocker therapy at a low dose under supervision.

Note 3: Food allergies rarely trigger acute asthma; however, a confirmed food allergy is a risk factor for asthma-related death.

Education and skills training

It is important to provide the parents or carers of children with asthma with information about the natural history of asthma, the rationale for treatment, and the need for good adherence with preventer medication (when prescribed).

Clearly explain the difference between preventer and reliever therapy to children and their carers.

A spacer device is vital for the delivery of all asthma drugs administered via pressurised metered dose inhaler (MDI) in children. Carefully demonstrate the appropriate spacer technique to parent or carer and the child.

A written asthma action plan is another important component of management.

If asthma symptom control is poor despite apparently adequate treatment, consider poor inhaler technique and/or poor adherence.

Regular review is important to ensure optimal control of symptoms with the lowest effective medication dose. Check adherence and device technique at each visit and perform spirometry if the child is capable. Consider stepping down treatment in children with good symptom control in the previous 3 months.

Update the written asthma action plan at least yearly or when maintenance treatment is changed.

Provide information on avoiding triggers, where appropriate, and managing comorbid conditions (eg allergic rhinitis). Advise on avoiding environmental tobacco smoke, and encourage physical activity, maintaining a healthy weight and a healthy lifestyle.

Failure to respond to adequate treatment

Most children with asthma have intermittent symptoms and do not require treatment beyond Step 1 of the stepwise approach, see above. Those who do require preventive treatment usually respond very well to first-line preventer therapy (ie Step 2 in figure above). Failure to respond to optimised first-line preventer treatment (ie correct technique and good adherence) should prompt reassessment of the diagnosis, and exploration of possible complicating factors, such as:

- exposure to aeroallergens or environmental and tobacco smoke
- psychosocial factors including parent or carer mental health problems, or financial hardship.

Referral may be warranted at this point; see below for further information about when specialist paediatric consultation is recommended.

Referral

Refer children for specialist paediatric consultation when there has been:

- a life-threatening asthma flare-up or asthma in conjunction with anaphylaxis
- frequent flare-ups requiring oral corticosteroids
- doubt about the diagnosis of asthma
- failure to respond to therapy, indicated by

- persistently impaired lung function (in children old enough to perform spirometry) despite control of symptoms
- the need for infants or toddlers to use oral or inhaled corticosteroids (ICS)
- the need for older children to use maintenance ICS doses in excess of the upper limit of the high-dose range.

Management of acute asthma in children 1 to 15 years

Overview

Acute asthma (also known as an asthma exacerbation, attack or flare up) is an acute worsening of lung function and asthma symptoms. Typical symptoms include shortness of breath, wheeze, cough and chest tightness.

Acute asthma usually occurs in response to a trigger, such as a viral respiratory tract infection or an irritant (eg pollen, pollution, cold air). Lack of adherence with preventer therapy is also a common factor.

This topic provides advice for managing acute asthma in children 1 to 15 years. For advice about managing acute asthma in adolescents, see asthma in adults and adolescents. Wheezing infants younger than 12 months old should not be treated for acute asthma. Acute wheezing in this age group is most commonly due to acute viral bronchiolitis.

In the event of an acute flare-up (exacerbation) of asthma, early intervention with inhaled bronchodilator therapy is the best strategy to prevent further deterioration.

Educate parents, carers and children with asthma to recognise early symptoms of deterioration and to initiate the first steps in treatment (see below).

Anaphylaxis and acute asthma

Anaphylaxis is an important differential diagnosis for children presenting with acute wheeze. Anaphylaxis is a life-threatening condition; the child can deteriorate exceedingly rapidly (ie within minutes).

Sudden-onset shortness of breath and typical skin features (eg any of urticarial rash, erythema, flushing or angioedema) is diagnostic of anaphylaxis. Anaphylaxis should also be considered if a patient presents with sudden-onset shortness of breath and cardiovascular symptoms (eg dizziness, hypotension) or gastrointestinal symptoms (eg diarrhoea, vomiting), even if typical skin features are not present.

If unsure if anaphylaxis or asthma, give empirical intramuscular adrenaline.

If anaphylaxis is suspected or cannot be excluded, give empirical intramuscular adrenaline (epinephrine):

adrenaline 10 micrograms/kg or 0.01 mL/kg of 1:1000 (maximum 0.5 mL) by IM injection into lateral thigh. Repeat after 5 minutes if the child is not improving.

First aid for acute asthma for patients and community members

If symptoms are **severe or life-threatening** urgently call an ambulance and advise that the child is having a 'severe asthma attack' (flare-up) and/or organise transport to hospital. Pending its arrival, administer high doses of inhaled short-acting beta$_2$ agonist (SABA) via pressurised metered dose inhaler (MDI) with spacer (if available) or via nebuliser.

In children younger than 6 years, use:

salbutamol 100 micrograms per puff (actuation), 6 separate puffs by inhalation via MDI with spacer, repeated every 20 minutes or sooner if required

OR

salbutamol 2.5 mg by inhalation via nebuliser, repeated every 20 minutes or sooner if required.

In children 6 years or older, use:

salbutamol 100 micrograms per puff (actuation), 12 separate puffs by inhalation via MDI with spacer, repeated every 20 minutes or sooner if required

OR

salbutamol 5 mg by inhalation via nebuliser, repeated every 20 minutes or sooner if required.

If early signs of asthma deterioration are present, first-line treatment with a bronchodilator should be initiated by the child, parent, or carer. Call an ambulance if the child fails to improve significantly with first-line home treatment, or if there are signs of a severe or life-threatening flare-up. Urgent medical review is also required if the child initially responds but bronchodilator therapy is needed more than 3 to 4-hourly.

Transfer patient to a higher level of care immediately if there are signs of a severe or life-threatening flare-up or if the child fails to respond rapidly to first-line treatment.

There is a '**first aid plan**' for acute asthma, used by many community and sports organisations, called '4×4×4', which recommends salbutamol MDI; if available, use:

- 4 separate puffs, with spacer if available, one puff at a time
- take 4 breaths from the spacer after each puff

- wait 4 minutes and then give another 4 separate puffs
- if the child still cannot breathe normally, call an ambulance, and continue giving 4 separate puffs every 4 minutes until the ambulance arrives.

Medical management of acute asthma in children

Overview

When the patient arrives at the medical facility, perform a rapid assessment to evaluate the severity of the flare-up, which determines initial management.

Most cases of acute asthma in children are mild and can be successfully managed in the primary care setting. Treat these children as for mild–moderate acute asthma as outlined above, with ongoing assessment of severity and response to treatment.

If the child's condition worsens, arrange urgent transfer to a divisional hospital.

The aim of therapy is to stabilise the child within the first hour, then attempt to lengthen the interval between salbutamol doses in a stepwise fashion.

Assessment

History

Inquire specifically about the duration and nature of symptoms, treatments used (relievers, preventers), trigger factors (including upper respiratory tract infection, allergy, passive smoking), pattern and course of previous acute episodes (eg. admission or ICU admissions), parental understanding of the treatment of acute episodes, and the presence of interval symptoms (see long term asthma control below).

Risk factors for severe disease

- previous ICU admission
- poor compliance to asthma therapy
- poorly controlled - significant interval symptoms
- past history of anaphylaxis

Examination

Wheeze is not a good marker of severity. The most important parameters in the assessment of the severity of acute childhood asthma are general appearance/ mental state and work of breathing (accessory muscle use, recession), as indicated in the table. Initial oxygen saturation (SpO2) on room air, heart rate and ability to talk are

helpful but less reliable additional features. Wheeze intensity, pulsus paradoxus and peak expiratory flow rate are not reliable.

Asymmetry on auscultation is often found due to mucous plugging, but warrants consideration of foreign body.

Children with respiratory distress should have minimal handling (see below).

Oxygen may be required for low saturations (SpO2 less than 90%), **do not** give for wheeze or increased work of breathing. The arterial oxygen saturation (SaO2) may be reduced in the absence of significant airway obstruction due to factors such as atelectasis and mucous plugging of airways. SaO2 is purely a measure of oxygenation, which may be preserved in the presence of deteriorating ventilation (with CO2 retention).

Tachycardia can be a sign of severity—but is also a side effect of beta agonists such as salbutamol.

Wheezing is an unreliable indicator of the severity of an asthma flare-up in children.

Wheezing may be absent in a severe flare-up (ie 'silent chest'). Cyanosis is only visible with marked hypoxaemia; it indicates life-threatening acute asthma, but its absence does not exclude life-threatening acute asthma (see Table 5).

Box 1: Minimal handling

The sick child deteriorates with handling and distressing procedures. Increased distress in an unwell child can:

- increase heart rate, respiratory rate and blood pressure
- cause de-oxygenation (especially in neonates)
- tip a child's condition from moderate to severe

Principles of minimal handling

- keep the child with parent or care giver
- keep the environment quiet and moderate lighting where possible
- allow the child comfort feeds if safe to do so
- minimise interventions, including examination and investigations that are not going to impact acute management
- group cares - eg observations and oral medications
- use comfort techniques for painful procedures such as intravenous catheters – eg local anaesthetic cream, distraction.
- do not forcibly alter a child's posture - especially in respiratory conditions such as croup. Children will naturally adopt the posture that facilitates the least airway obstruction.

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Investigations

Chest X-ray is not generally required (discuss with senior doctor if considering). Arterial blood gas and spirometry are **not** required in the assessment of acute asthma in children.

Blood gases are distressing and can cause a child with respiratory compromise to deteriorate further. They are not usually required; the child's clinical state is more important in guiding therapy.

The most important parameters of severity in acute childhood asthma are the child's general appearance or mental state, and work of breathing (eg accessory muscles use, chest wall recession).

Treatment of acute asthma in children is based on assessment of severity

Assess severity of the acute asthma episode (moderate, severe or life-threatening) and administer a bronchodilator immediately:

- Make a rapid clinical assessment with the child in a sitting position
- Measure pulse oximetry while the person is breathing air (unless life threatening)
- Start bronchidilator therapy according to severity and age

Table 8: Initial rapid severity assessment of acute asthma in children

If features of more than one severity category are present, record the higher (worse) category as the overall severity level.

Mild to moderate: **all of the following**	**Severe:** **any of the following**	**Life-threatening:** **any of the following**
• can walk and speak whole sentences in one breath (for young children; can move around, speak in phrases) • SpO_2 more than 94%	• unable to complete sentences in one breath • increased work of breathing with use of accessory muscles (eg tracheal tug, intercostal or subcostal recession, marked abdominal breathing, chest wall recession in children) • obvious respiratory distress • Sp02 90 to 94%	• reduced consciousness • collapse • exhaustion • cyanosis • poor respiratory effort • soft or absent breath sounds • SpO_2 less than 90%

cont...

Important additional information:

- The severity category may change when more information is available or over time; ongoing observation is required.
- If oxygen therapy has already been started, it is not essential to stop oxygen to monitor oximetry.
- Oxygen saturation levels are a guide only; clinical judgement should be applied.
- While clinical features can help identify severity of an acute asthma flare-up, they are not specific (either in isolation or in combination); their absence does not exlude a severe or life-threatening asthma flare-up. Of note:
  - wheezing may be absent in severe acute asthma (ie 'silent chest')
  - pulsus paraoxus is not a reliable indicator of the severity of acute asthma
  - life-threatening acute asthma can occur without cyanosis.

SpO_2 = oxygen saturation measured by pulse oximetry

Adapted from the *Australian Asthma Handbook* © 2020 National Asthma Council Australia. Accessed March 2022.

Management

Consider consultation with the paediatric team if any of the following:

- assessed as moderate or severe asthma
- poor response to inhaled salbutamol
- oxygen requirement

Consider transfer to a divisional hospital if any of the following:

- severe or critical asthma requiring intravenous treatment or respiratory support
- children with escalating oxygen requirement
- children poorly responsive to salbutamol or unable to wean salbutamol
- children requiring care above the level of comfort of the local hospital.

Management of mild to moderate acute asthma in children

can walk and speak whole sentences in one breath

SpO2 more than 94%

For a patient with moderate acute asthma according to the initial rapid assessment, start treatment with salbutamol. Use:

salbutamol 100 micrograms per puff (actuation), 1 puff at a time via MDI with spacer

child 1 to 5 years: 6 puffs every 20 minutes for 1 hour (or sooner if needed)

child 6 years or older: 12 puffs, every 20 minutes for 1 hour (or sooner if needed)

OR

salbutamol via intermittent nebulisation

child 1 to 5 years:2.5 mg every 20 minutes for the first hour (or sooner if needed)

child 6 years or older: 5 mg every 20 minutes for the first hour (or sooner if needed).

Review the child 10 to 20 minutes after third dose to decide on timing of next dose.

Consider **oral prednisolone** (see below):

prednisolone 2 mg/kg (maximum 60 mg) orally for the initial dose, only continuing with 1 mg/kg orally, once daily for a further 1-2 days if there is ongoing need for regular salbutamol.

Management of severe acute asthma in children

unable to complete sentences in one breath
increased work of breathing with use of accessory muscles (eg tracheal tug, intercostal or subcostal recession, marked abdominal breathing, chest wall recession in children)
obvious respiratory distress
Sp02 90 to 94%

If the patient is being managed in primary care, arrange urgent transfer to a divisional hospital.

Admit any child with severe acute asthma to ICU and involve a consultant in their care.

If oxygen saturation measured by pulse oximetry (Sp02) is less than 92%, start supplemental oxygen therapy. Titrate oxygen to a target Sp02 of 92 to 96%; need for oxygen should be reassessed regularly. Do not give oxygen for wheeze or increased work of breathing. For more detailed advice on supplemental oxygen administration, see 'Acute oxygen therapy' page 161.

If a child is deteriorating at any stage, treat as critical asthma.

For a child with severe acute asthma according to the initial rapid assessment, start treatment with **both** salbutamol and ipratropium.

salbutamol 100 micrograms per puff (actuation), 1 puff at a time via MDI with spacer

child 1 to 5 years: 6 puffs every 20 minutes for 1 hour (or sooner if needed)

child 6 years or older: 12 puffs, every 20 minutes for 1 hour (or sooner if needed)

OR

salbutamol via intermittent nebulisation

child 1 to 5 years:2.5 mg every 20 minutes for the first hour (or sooner if needed)

child 6 years or older: 5 mg every 20 minutes for the first hour (or sooner if needed)

Review ongoing requirements for salbutamol 10-20 minutes after the third dose. If clinically improving, reduce frequency. If no change, continue to give every 20 minutes.

PLUS

ipratropium bromide via intermittent nebulisation every 20 minutes for 1 hour for 3 doses only

child 1 to 5 years: 250 micrograms per dose

child 6 years or older: 500 micrograms per dose

PLUS

hydrocortisone 4 mg/kg (up to 100 mg) intravenously, 6-hourly for up to 24 hours; switch to an oral corticosteroid once tolerated. If intravenous therapy is still required after 24 hours, reduce frequency to 12-hourly

Consider adrenaline if the child is not improving:

adrenaline (epinephrine) 1 mg/mL (1:1000, 0.1%) solution, 0.01 mg/kg up to 0.5 mg (0.5 mL) intramuscularly; repeat after 3 to 5 minutes if required.

Arrange admission after initial assessment and management.

Management of critical / life-threatening acute asthma in children

reduced conciousness
collapse
soft or absent breath sounds
poor respiratory effort
exhaustion
cyanosis
SpOo2 less than 90%

Consider anaphylaxis as a differential diagnosis—see 'Anaphylaxis and acute asthma' for more information.

Call for assistance – request urgent involvement from a consultant or senior PICU registrar.

For a patient with life-threatening acute asthma according to the initial rapid assessment, arrange immediate transfer to a critical care or high-dependency facility. Early involvement of senior staff is required.

Check doses of intravenous medicines carefully – see below for additional information on dosing and administration

Supplemental oxygen is almost always required for patients with life-threatening acute asthma. Use high flow via oxygen mask (15 L/minute) and titrate oxygen to a target SpO2 of 92 to 96%.

Start immediate treatment with nebulised bronchodilator therapy (both salbutamol and ipratropium). Use oxygen to drive the nebuliser:

salbutamol via continuous nebulisation

child 1 to 5 years:2.5 mg at a time, undiluted

child 6 years or older: 10 mg at a time, undiluted

PLUS

ipratropium bromide via nebulisation, added to salbutamol, every 20 minutes for 3 doses then every 4 hours

child 1 to 5 years: 250 micrograms per dose

child 6 years or older: 500 micrograms per dose

Give intravenous corticosteroid therapy as soon as possible (and at least within the first hour):

hydrocortisone 4 mg/kg (up to 100 mg) intravenously, 6-hourly for up to 24 hours; switch to an oral corticosteroid once tolerated. If intravenous therapy is still required after 24 hours, reduce frequency to 12-hourly

If the patient worsens or does not have a rapid and marked response to bronchodilator and oxygen therapy, add intravenous magnesium sulfate (not suitable for children younger than 2 years). Use:

magnesium sulfate 10 mmol (2.5 g/5 mL; 50%) (child 2 years or older: 0.1 mmol/kg up to 8 mmol (equivalent to approximately 25 mg/kg up to 2 g) diluted to 50 mL in normal saline, infused intravenously via syringe pump over 20 minutes

Higher doses are sometimes used – check with a consultant

In an ICU setting, this can be continued:

magnesium sulfate 10 mmol (2.5 g/5 mL; 50%) (child 2 years or older: 0.12 mmol/kg/hour infused intravenously via syringe pump.

Comprehensive monitoring in a critical care or high-dependency environment is required.

Consider intravenous salbutamol:

salbutamol 10 micrograms/kg (maximum 500 micrograms) intravenously as a single bolus dose given over at least 2 minutes. Consider repeating dose at 10 minutes if not improving.

Comprehensive monitoring (blood electrolytes, heart rate, blood lactate) in a critical care or high-dependency environment is required.

Consider intravenous aminophylline:

aminophylline loading dose (omit in children being treated with oral theophylline) 10 mg/kg (maximum 500 mg) intravenously over 1 hour.

If inadequate response, start a continuous infusion (in PICU setting only):

aminophylline intravenous infusion via syringe pump

child 1 to 9 years: 1.1 mg/kg/hour

child 10 to 15 years: 0.7 mg/kg/hour.

Comprehensive monitoring in a critical care environment, including cardiac monitoring, is required.

Treatment in children is often complicated by nausea and vomiting. Theophylline plasma concentrations should be monitored to ensure they are within the therapeutic range and the dose adjusted accordingly, however this is currently unavailable in Fiji.

Note: aminophylline, magnesium and salbutamol must be given via separate IV lines

Additional treatment for persistent life-threatening acute asthma

Adrenaline (epinephrine)

If the patient is unresponsive, has poor respiratory effort, and cannot inhale bronchodilators, or is considered to be peri-arrest, consider adrenaline (epinephrine). Give:

adrenaline (epinephrine) 1 mg/mL (1:1000, 0.1%) solution, 0.01 mg/kg up to 0.5 mg (0.5 mL) intramuscularly; repeat after 3 to 5 minutes if required.

Monitoring in severe and critical / life-threatening acute asthma

Children with severe or critical / life-threatening asthma should be managed by a consultant in a critical care or high dependency environment.

Monitoring required:

- continuous cardiac monitoring
- non-invasive blood pressure, oxygen saturation and respiratory rate
- blood electrolytes, including magnesium and calcium, and blood lactate
- capullary blood gas (CBGs) and areterial blood gas (ABGs) at baseline and then at least 6-hourly

Salbutamol toxicity: tachycardia, tachypnoea, metabolic acidosis; can occur with both intravenous and inhaled treatment. Lactate levels are commonly high. Hypokalaemia and hypomagnesaemia are expected when repeated or high doses are given and should be managed appropriately. The effects on the cardiovascular system may lead to adverse consequences such as myocardial ischaemia or prolonged QT interval; the latter predisposes to arrhythmias, especially in the context of electrolyte disturbances.

High doses of salbutamol may paradoxically worsen the respiratory compromise. This effect is thought to be multifactorial; metabolic acidosis, rise in lactate levels and increased metabolic rate may all contribute. If suspected, the patient should be closely observed and salbutamol treatment should be cautiously back-titrated; specialist input is recommended.

The safety of repeated doses of magnesium sulfate has not been assessed. Hypermagnesaemia may cause loss of deep tendon reflexes and muscle weakness, including respiratory muscle weakness.

The potential harm versus benefit of intravenous aminophylline is considered unfavourable in the majority of situations. It can cause vomiting, arrhythmias, convulsions and sudden death, and is rarely used. Aminophylline interacts with many medicines, including antibiotics such as erythromycin and ciprofloxacin. Check current medications before starting treatment.

See the PICU Clinical Practice Guidelines for further information on management of life-threatening asthma, including ventilatatory support.

Corticosteroids in the management of acute asthma in children

Corticosteroid therapy hastens symptom resolution and prevents relapse in acute asthma. It is recommended for:

- all cases of acute asthma in children 6 years or older, except the mildest of cases (eg mild symptoms that respond quickly and completely to bronchodilator therapy)

- all cases of severe acute wheezing in children 1 to 5 years; in a child with only a mild to moderate wheeze corticosteroids may be of no benefit so can be withheld to reduce corticosteroid exposure.

Discharge and follow-up

Discharge criteria:

- one hour after initial assessment, if reassessed as mild severity and no risk factors for severe disease
- adequate oxygenation: mild hypoxia (Sp02 90-94) should not preclude discharge if child is clinically well and has responded well to treatment
- parental education provided and ability to administer salbutamol via spacer checked
- adequate oral intake
- adequate supply of reliever medication at home

Discharge information requirements:

- give a new written asthma action plan
- educate on reliever use
- observe inhaler technique before discharge.
- advise parents to seek further medical attention should the patient's condition deteriorate or if there is no significant improvement within 48 hours.
- at discharge, all patients should have an outpatient appointment or appropriate follow-up arranged with a GP and/or paediatrician.
- parents should be informed of other sources of information about asthma eg from the Australian National Asthma Foundation <https://www.nationalasthma.org.au/living-with-asthma/resources/patients-carers>

Asthma in adults and adolescents

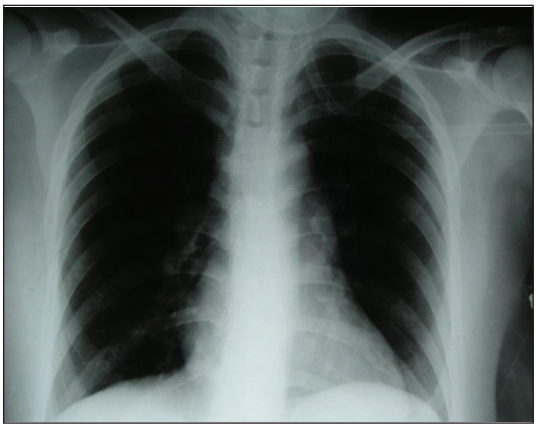
Introduction

This topic addresses the diagnosis and management of asthma in adults and adolescents; for advice about children 15 years and younger, see 'Asthma in children.'

Asthma is described as a 'chronic lung disease, which can be controlled but not cured.' Its major characteristics include a variable degree of airflow obstruction, hyperresponsiveness and airway inflammation. Asthma is a common disease, diagnosed clinically, with variable clinical presentations at different times over a patient's lifetime. Appropriate recognition of disease activity and optimisation of acute and longer-term maintenance care is challenging.

In overseas countries, the introduction of a variety of new drugs and devices offers potential for more appropriately tailored care but at the risk of patient and clinical confusion.

Image 1: An example of an X-ray image in asthma



Diagnosis

General information

Asthma frequently presents in childhood but can occur for the first time at any age. There are concerns about both over- and under-diagnosis of asthma in children and adults.

There is no single diagnostic test for asthma.

The clinical diagnosis in adults is made by:

- considering asthma as a potential diagnosis for a variety of respiratory symptoms including wheeze, dyspnoea (shortness of breath), cough and chest discomfort or tightness
- taking a detailed history to identify the pattern of symptoms and to exclude other causes
- documenting variable airflow limitation.

Asthma is characterised by respiratory symptoms (wheeze, shortness of breath, chest tightness, cough) that vary in intensity and over time. A summary of the key symptom patterns in adults and adolescents consistent with asthma is provided in the table below.

In older people, key alternative diagnoses to exclude are chronic obstructive pulmonary disease (COPD), left ventricular failure, bronchiectasis and lung cancer. Asthma and COPD may coexist in older patients and awareness of this reduces the likelihood of mismanagement (particularly the use of inappropriate medications).

Table 9: Diagnosis of asthma in adults and adolescents

CLINICAL FEATURES THAT INCREASE THE PROBABILITY OF ASTHMA
• more than one of the following symptoms: wheeze, breathlessness, chest tightness or discomfort, cough particularly if symptoms: – are worse at night and in the early morning – occur in response to exercise, allergen exposure or cold air – occur after taking aspirin or beta blockers – are recurrent • history of atopic disorder, eg allergic rhinitis, atopic dermatitis • family history of asthma and/or atopic disorder • widespread wheeze heard on auscultation of the chest • improvement in symptoms of lung function in response to standard asthma therapy • otherwise unexplained low FEV_1 or PEF (historical or serial readings) • otherwise unexplained peripheral blood eosinophilia.

cont...

CLINICAL FEATURES THAT LOWER THE PROBABILITY OF ASTHMA

- chronic productive cough in the absence of wheeze or breathlessness
- normal FEV_1 or PEF when symptomatic [Note 1]
- repeatedly normal ascultation of chest when symptomatic
- voice disturbance or throat tightness
- symptoms that worsen with talking or laughing
- prominent dizziness, light-headedness, peripheral tingling
- symptoms that only occur with viral respiratory infections, with few or no symptoms in between
- no response to a trial of asthma therapy
- significant smoking history (more than 20 pack years [Note 2])
- clinical features supporting an alternative diagnosis

Record the basis on which a diagnosis of asthma is suspected.

FEV_1 = forced expiratory volume in 1 second; PEF = peak expiratory flow

Note 1: Normal spirometry when not symptomatic does not exclude the diagnosis of asthma; ideally, repeat spirometry when the patient is symptomatic. If spirometry is normal when the patient is symptomatic, consider an alternative diagnosis. Repeated measurements of lung function are often more informative than a single assessment.

Note 2: Pack years is calculated using the formula (years of smoking × cigarettes per day) / 20. A significant smoking history could indicate that the patient has chronic obstructive pulmonary disease (COPD). Note that asthma and COPD can coexist.

Table 10: Alternative diagnoses other than asthma to consider in adults and adolescents according to predominant symptoms

Symptom	**Possible alternative diagnosis** [Note 1]
breathlessness	lack of fitness
	obesity
	COPD
	hyperventilation or dysfunctional breathing
	upper airway dysfunction (vocal cord dysfunction)
	pleural effusion
	pulmonary fibrosis
	lung cancer
	large airway stenosis
	inhaled foreign body
	pulmonary hypertension
	congenital heart disease
	heart failure
wheeze	COPD
	Bronchiectasis
	Acute Bronchitis
	upper airway dysfunction (vocal cord dysfunction)
	large airway stenosis
	large airway malacia
	inhaled foreign body
	Heart Failure
chest tightness	ischaemic heart disease
	hyperventilation or dysfunctional breathing
	gastro-oesophageal reflux

cont...

dry cough	postviral cough
	upper airway cough syndrome (postnasal drip)
	oesophageal disorders (eg gastro-oesophageal reflux)
	drug-induced (eg ACEI)
	COPD
	chronic rhinosinusitis
	lung cancer
	inhaled foreign body
	pulmonary fibrosis
sputum production	COPD
	chronic bronchitis
	bronchiectasis
	rhinitis
	lung cancer
	cystic fibrosis
	inhaled foreign body

ACEI = angiotensin converting enzyme inhibitor; COPD = chronic obstructive pulmonary disease

Note 1: Likelihood of alternative diagnoses will depend on other patient-specific factors such as age, comorbid conditions, smoking history and other findings.

Variable airflow limitation

Evidence of variable expiratory airflow limitation should be obtained and documented in the patient's clinical records. This should preferably be done before starting regular preventer treatment, as it is harder to confirm the diagnosis of asthma once the patient is receiving treatment. In urgent asthma presentations, empirical treatment with inhaled corticosteroids may be started, and the patient brought back for objective testing within 1 to 3 months.

In some patients, observing a response after 1 to 3 months of regular preventer treatment may help to confirm the diagnosis, but lack of response to bronchodilators or inhaled corticosteroids does not rule out asthma.

The term 'variable airflow limitation' is used when the variation in expiratory airflow is more than that seen in healthy people. However, there is some variation in lung function even in healthy people and patients with chronic obstructive pulmonary disease (COPD) (up to 12% from week to week). In a patient with symptom patterns consistent with asthma, the more times variable airflow limitation is seen, or the greater the variation, the more likely the diagnosis of asthma.

Criteria for variable airflow limitation include:

- clinically important bronchodilator reversibility; ie an increase of forced expiratory volume in 1 second (FEV1) of at least 200 mL and 12% from baseline 10 to 15 minutes after giving a short-acting beta$_2$ agonist (SABA) (200 to 400 micrograms inhaled salbutamol). A larger increase in FEV1 (ie more than 400 mL) in response to a SABA is strongly suggestive of asthma
- clinically important variation in lung function (at least 20% change in FEV1 or peak expiratory flow [PEF]) when measured repeatedly over time
- diurnal variation in PEF readings of greater than 10% [Calculated each day from twice daily PEF readings (best of three each time) as ([the day's highest PEF minus the day's lowest PEF] divided by the mean of the day's PEF) and averaged over 1 to 2 weeks]. Use the same meter on each occasion
- positive bronchial provocation challenge or exercise challenge in a respiratory function laboratory
- clinically important increase in FEV1 of more than 200 mL and 12% from baseline after a therapeutic trial of inhaled corticosteroids for 4 weeks.

At least once during the diagnostic process, confirm the presence of expiratory airflow limitation (ie reduced FEV1 to forced vital capacity [FVC] ratio [FEV1/FVC]). In most healthy adults, the FEV1/FVC is greater than 0.75 to 0.8 (sometimes expressed as a percentage [75 to 80%]) and for adolescents, greater than 0.85 (85%). In older people, an FEV1/FVC as low as 0.70 (70%) may still be considered normal.

Normal spirometry in the absence of symptoms does not exclude asthma. However, if the patient is symptomatic (especially if short of breath) at a time when spirometry is normal, consider another diagnosis. The clinical diagnosis should also be reviewed if management is ineffective.

Review the clinical diagnosis if management is ineffective.

Maintenance management of asthma in adults and adolescents

Overview

The long-term goals of asthma management are:

- symptom control; ie to achieve good control of symptoms and maintain normal activity levels
- risk reduction, ie to minimise future risk of
  - flare-ups (exacerbations)
  - fixed airflow limitation
  - drug adverse effects.

These goals are achieved through:

- accurate diagnosis
- a partnership between the patient and healthcare professional(s)
- control-based management, incorporating
  - drug treatment, managed with a cycle of assessment, adjustment and review of response
  - nondrug interventions
  - management of modifiable risk factors and comorbid conditions
- education and skills training for asthma self-management
- regular medical review.

Doctor–patient relationship

Effective asthma care requires a partnership between the patient and healthcare professional(s) so that patients can participate in decisions about their management and gain confidence and skills to engage in self-management. Patients vary in their willingness and ability to participate in self-management.

Good communication is an essential part of asthma management. Take the patient's health literacy into account when providing education and skills training. Specific communication strategies can assist in reducing the impact of low health literacy, eg ordering information from the most to the least important, and using the 'teach-back' method to confirm understanding.

Control-based management

Overview

The aim of asthma management is to achieve good long-term asthma control. Asthma control refers to the extent to which the manifestations of asthma have been reduced or removed by treatment; it has two components:

- current level of symptom control
- risk of future adverse outcomes.

Assessing asthma symptom control is different to assessing asthma severity.

Assessing asthma symptom control

Good asthma symptom control is characterised by:

- absent or minimal daytime symptoms
- no nocturnal symptoms
- no limitation of activity by asthma
- minimal need for short-acting $beta_2$ agonists.

The patient's **current level of symptom control** can be summarised with a validated composite score, eg Asthma Control Test (referred to as the Asthma Score in Australia; see <www.asthmaaustralia.org.au/AsthmaScore>). The Primary care Asthma Control Screening tool (PACS) (see Australian Asthma Handbook <www.asthmahandbook.org.au>) can be used to quickly identify patients who need more detailed assessment of their asthma symptom control.

In some patients, symptoms and reliever use may be an unreliable indicator of the need for regular preventer therapy. The following situations highlight the need to measure lung function as well as assess symptoms and reliever use.

Symptoms and reliever use may be inappropriately infrequent, leading to undertreatment, in patients who are:

- inactive, ie with insufficient activity to experience symptoms
- poor perceivers of airway obstruction—consider if a substantial change in lung function (eg 20% increase after bronchodilator or similar decrease after exercise or during bronchial provocation testing) is not accompanied by a change in symptoms
- using a long-acting bronchodilator alone without inhaled corticosteroid (ICS). Using long-acting $beta_2$ agonists (LABAs) alone is dangerous and not recommended; patients may have few symptoms but are at high risk of flare-ups and an increased risk of asthma-related death due to uncontrolled airway inflammation.

Symptoms and reliever use may be inappropriately frequent, leading to overtreatment and risk of adverse effects, in patients who are:

- experiencing symptoms due to conditions other than asthma, eg shortness of breath due to obesity, or cough due to chronic upper airway cough syndrome (postnasal drip)
- overperceivers of airway obstruction
- using excessive doses of short-acting $beta_2$ agonist through habit or anxiety.

Assessing risk of adverse asthma outcomes

Assess patients for their risk of future adverse outcomes including flare-ups, faster than normal decline in lung function and adverse effects of treatment.

Patients are at risk of adverse asthma outcomes, even if they have few asthma symptoms, if they:

- have poor lung function
- smoke or are exposed to environmental tobacco smoke
- are using too much or too little of their prescribed therapy
- are using high doses of ICS.

Other factors predictive of worse clinical outcomes include increased airway hyperresponsiveness and blood eosinophilia.

A more extensive list of adverse asthma outcomes and associated risk factors is provided in the table below. Review patients at risk of adverse outcomes more frequently than usual.

Table 11: Risk factors for adverse asthma outcomes in adults and adolescents

Risk factors for exacerbations
poor adherence
inadequate inhaler technique
poor asthma symptom control
any asthma exacerbation during the previous 12 months
lack of written asthma action plan
difficulty perceiving airflow limitation or severity of exacerbation
poor lung function (even if few symptoms)
peripheral blood eosinophilia (suggests eosinophilic airway inflammation)
exposure to tobacco smoke (personal or second-hand smoking)
socioeconomic disadvantage
use of illegal substances
major psychosocial problems
mental illness
other chronic lung disease
high bronchodilator reversibility
allergic rhinitis or rhinosinusitis
obesity

cont...

Risk factors for life-threatening asthma
intubation or admission to intensive care unit because of asthma (ever)
two or more hospitalisations for asthma in the past year
three or more ED visits for asthma in the past year
hospitalisation or ED visit for asthma in the past month
high SABA use (use of three or more canisters per year is associated with increased risk of exacerbation; use of more than 12 canisters per year is associated with increased risk of asthma-related death)
history of delayed presentation to hospital during exacerbations
history of sudden-onset acute asthma
lack of written asthma action plan
sensitivity to an unavoidable allergen (eg *Alternaria* species of common moulds)
confirmed food allergy
inadequate treatment
experience of adverse effects of oral corticosteroids (may contribute to under treatment or delayed presentation to hospital during exacerbations)
socioeconomic disadvantage
living alone
mental illness
use of alcohol or illegal substances
poor access to health care (eg rural or remote region)
cardiovascular disease
Risk factors for accelerated decline in lung function
chronic mucus hypersecretion
severe asthma exacerbation in a patient not using ICS
occupational asthma
poor lung function
peripheral blood eosinophilia (suggests eosinophilic airway inflammation)
exposure to tobacco smoke (personal or second-hand smoking)

cont...

Risk factors for treatment-related adverse events
long-term high-dose ICS
frequent use of oral corticosteroids
anxiety disorder (may be associated with increased sensitivity to asthma symptoms and reluctance to reduce ICS dose when asthma is well controlled)
euphoria with oral corticosteroid use

ED = emergency department; ICS = inhaled corticosteroid; SABA = short-acting beta$_2$ agonist.

Adapted from the *Australian Asthma Handbook* © 2020 National Asthma Council Australia. Accessed March 2022.

Assessing asthma severity

Asthma severity is defined by the intensity of treatment required to achieve good asthma symptom control. It is a retrospective label that is applied after a patient has been using preventer treatment for at least 3 to 6 months.

Asthma severity is defined by the intensity of treatment required to achieve good asthma symptom control.

Note that the term 'severity' may also be applied to the severity of an acute flare-up.

Mild asthma is asthma that can be well controlled with occasional short-acting beta$_2$ agonist alone or with low-dose inhaled corticosteroid; ie Step 1 or 2 in the figure below.

Severe asthma is asthma that requires high-intensity treatment (high-dose inhaled corticosteroid plus long acting beta$_2$ agoinst, with or without other therapy) to maintain good control, or when good control is not achieved despite high-intensity treatment; ie Step 4 or higher in the figure below.

It is possible for severe asthma to be well controlled because the patient is using optimal doses of medication; it is also possible for mild asthma to be poorly controlled because the patient is not using sufficient preventer medication.

It is important to achieve good asthma symptom control. Maintain this for 3 months, then step-down to find the minimum effective treatment which is then used as a guide to the severity of the patient's asthma. Review the severity and treatment at least annually.

Asthma can be difficult to treat in some patients as it can be challenging to completely resolve problems such as poor adherence, poor inhaler technique, and comorbid conditions.

Stepwise approach to maintenance drug treatment

Overview

Specific strategies for asthma maintenance treatment include:

- drug treatment—inhaled, oral and parenteral
- nondrug interventions
- managing modifiable risk factors including triggers and common comorbid conditions.

Route of administration

Inhalation is the preferred route of drug administration in asthma. Inhalation allows deposition of the drug into the bronchial tree, resulting in the lowest effective dose and minimal systemic adverse effects. Inhaled drugs can be administered by pressurised metered dose inhalers (MDIs) or dry powder inhalers (DPIs [not available in Fiji]). Nebulisers are only rarely needed. Effective administration of inhaled drugs requires correct delivery.

When different devices are available, the device and regimen depends firstly on the drug that meets the patient's clinical needs; appropriate device choice is then based on various factors including patient preference and capabilities, age, and susceptibility to local adverse effects.

Less complicated treatment regimens can improve adherence to therapy.

Spacers should be used by adults and adolescents for using any MDI (especially for inhaled corticosteroid Inhalers). A spacer reduces problems with coordination, increases the relative dose delivered to the lung and decreases oropharyngeal deposition.

To minimise the risk of oropharyngeal candidiasis and systemic corticosteroid absorption, advise patients to rinse their mouth with water and spit out straight after using an ICS. Using a spacer with an ICS also reduces systemic absorption, oropharyngeal candidiasis and dysphonia.

Initiating treatment

Once current level of symptom control and risk factors are identified, and baseline lung function is documented, select appropriate initial treatment – see below. Some patients may have started self-treatment with an over-the-counter short-acting beta$_2$ agonist (SABA).

Choice of initial preventer treatment for adults and adolescents presenting with asthma depends on their recent symptom pattern. For patients presenting with:

- symptoms less than twice per month, who have not had a flare-up (exacerbation) requiring oral corticosteroids in the previous year, and without risk factors for exacerbations (see Table 11), start (or continue) treatment with as-needed SABA
- symptoms occurring at least twice per month, or who have woken due to asthma symptoms at least once in the past month, and/or who have had a flare-up requiring oral corticosteroids in the previous year, or have risk factors for exacerbations (see Table 11), start preventer treatment with regular low-dose inhaled corticosteroid (ICS), plus SABA as needed
- poorly controlled or very troublesome symptoms (eg frequent night waking, poor lung function) on initial presentation, start preventer treatment with medium- to high-dose ICS. A short course of oral corticosteroids may be considered. Start (or continue) as-needed SABA. Step-down the ICS dose after symptoms improve and good asthma symptom control (See previous Section on Assessing Asthma Symptom Control) is maintained for 2 to 3 months (see Figure 2).

Stepwise approach to treatment in adults and adolescents

Overview

Treatment aims to achieve symptom control as quickly as possible. In some cases this may involve starting with a higher-dose medication regimen and then stepping down treatment. Clearly advise the patient of the plan and the need for follow-up to achieve this.

Ongoing treatment aims to use the lowest dose of drugs that maintains asthma symptom control and prevents flare-ups. If inhaled corticosteroids (ICS) are indicated, low-dose regular treatment with ICS (plus short-acting beta$_2$ agonist [SABA] as needed) is more effective than intermittent treatment and more likely to achieve control with a lower total dose of ICS.

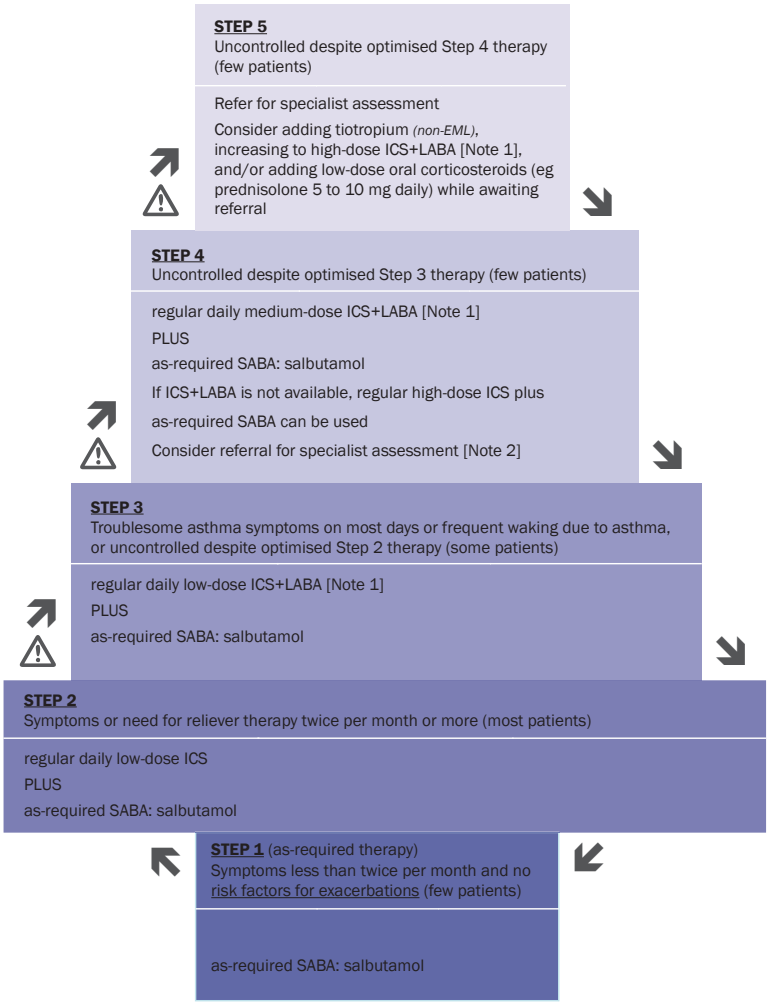
Low-dose regular ICS (inhaled corticosteroid) is more effective than intermittent treatment.

A stepwise approach to treating asthma in adults and adolescents is outlined below. The steps reflect the increased intensity of medication required to achieve good asthma symptom control for patients who have not responded to the previous level despite good adherence and correct inhaler technique.

Approximately three-quarters of adults with asthma have mild asthma that can be well controlled by Step 2 treatment.

Most adults with asthma have mild asthma that can be well controlled with low-dose ICS preventer treatment and as-needed SABA (short-acting beta$_2$ agonist).

Figure 2: Stepped approach to adjusting asthma medication in adults and adolescents



⚠	Before considering stepping up therapy, review adherence and inhaler technique, check equipment (eg inhaler, spacer, mask) for breakage or blockage, assess for an alternative diagnosis or a comorbidity (eg rhinitis), and ensure exposure to triggers is minimised.
↗	Consider stepping up therapy if good control is not achieved despite good adherence and correct inhaler technique; see Assessing asthma symptom control for more information.
↘	If asthma has been stable and well controlled for 2 to 3 months, consider stepping down therapy; see Stepping down therapy for more information.

cont...

Note 1: Always give ICS+LABA therapy as a fixed-dose combination inhaler to avoid the possibility of patients taking a LABA without an ICS; LABA monotherapy increases the risk of exacerbations and asthma-related death.

Note 2: Consider adding tiotropium while awaiting referral non-EML

Additional references: The Australian Asthma Handbook © National Asthma Council of Australia.

In the event that a LABA is not available and patients are still at Step 3 or Step 4, refer patients to the closest divisional hospital (Colonial War Memorial, Lautoka or Labasa Hospital) or an internal medicine specialist.

Long term oral steroids and theophylline should not be used without specialist consultation.

Stepping up treatment

After 1 to 3 months, if good asthma symptom control is not achieved, consider stepping up treatment only after confirming:

- symptoms are due to asthma
- inhaler technique is correct
- adherence is adequate.

Identify and manage any comorbid conditions that may be affecting asthma symptom control.

Stepping down treatment

Consider stepping down treatment if asthma is stable and well controlled for 2 to 3 months. Check the patient's actual treatment regimen before stepping down treatment, remembering that what the patient is using may not necessarily be what has been prescribed. Assess the patient's risk factors for flare-ups and choose an appropriate time to step-down. For example, do not step-down when the patient has a cold and not before the patient's access to medical services will be limited, such as on public holidays or when travelling internationally or to remote areas.

During pregnancy, therapy should not be stepped down due to the risk to mother and baby of any flare-ups.

If stepping down treatment is considered appropriate, either:

- reduce ICS dose (25 to 50% dose reduction every 2 to 3 months), or
- stop LABA if ICS dose is already low

Stopping ICS in adults is associated with a significant risk of asthma flare-up; if an adult has stopped ICS they should be made aware of this risk. Adults using low-dose ICS should generally not be stepped down to SABA alone; however, this may be warranted in some circumstances (eg confirming asthma diagnosis). In these cases, as with any step-down in treatment, assess the risk of flare-up, and closely monitor

and follow up the patient.

Following any step-down in treatment, advice patients to step back up if there is deterioration in control. Update the patient's written asthma action plan regularly during any change.

Drug doses and administration

Inhaled corticosteroids

Dosage

Inhaled corticosteroids (ICS) (beclomethasone) are first-line maintenance treatment. They are referred to as 'preventers'.

Start with low-dose ICS in addition to as-needed short-acting beta$_2$ agonist (SABA); use:

a low-dose ICS by inhalation, see Table 12 below.

Table 12: Corticosteroid-based-inhalers available in Fiji for asthma in adults and adolescents

Drug	**Dosage** [Note 1]		
	Low	**Medium**	**High [Note 2] [Note 3]**
beclometasone	50 or 100 micrograms twice daily	200 micrograms twice daily	300 or 400 micrograms twice daily
budesonide (including combinations with LABA) Non-EML	100 or 200 micrograms twice daily	400 micrograms twice daily	600 or 800 micrograms twice daily maximum 2400 micrograms daily in divided doses
fluticasone propionate (including combinations with LABA) Non-EML	50 or 100 micrograms twice daily	125 to 250 micrograms twice daily	500 micrograms twice daily maximum 1000 micrograms twice daily

cont...

COPD = chronic obstructive pulmonary disease; DPI = dry powder inhaler; LAMA = long-acting muscarinic antagonist; SABA = short-acting beta$_2$ agonist; MDI = pressurised metered dose inhaler

Note 1: For information about delivery devices, including links to videos and patient handouts, see 'Inhalational drug delivery devices.'

Note 2: Unless a maximum daily dose is specified, the high-dose regimen is also the maximum daily dose.

Note 3: Except for short periods, refer patients considered for high doses to a specialist.

Non-corticosteroid-based inhalers

Table 13: Non-corticosteroid-based inhalers available in Fiji for asthma in adults and adolescents

[Note 1]

Drug	Device [Note 2]	Dose per inhalation
SABA inhalers		
Salbutamol	MDI	100 micrograms
LAMA inhalers		
tiotropium Non-EML	mist inhaler	2.5 micrograms
	DPI	13 micrograms, 18 micrograms [Note 3] Tiotropium DPI is available as a 13 microgram inhaler and an 18 microgram inhaler. These inhalers are bioequivalent, both delivering 10 micrograms per dose, and can be used interchangeably.

COPD = chronic obstructive pulmonary disease; DPI = dry powder inhaler; LAMA = long-acting muscarinic antagonist; SABA = short-acting beta$_2$ agonist; MDI = pressurised metered dose inhaler

Note 1: Cromones (cromoglycate, nedocromil) were formerly used to treat asthma, but they are less effective than inhaled corticosteroids for controlling asthma and improving lung function, so are rarely used and are not available on the Fiji EML at the time of publication.

Note 2: For information about delivery devices, see Inhalational drug delivery devices page 197.

Note 3: Tiotropium via mist inhaler (but not DPI) is marketed as add-on maintenance treatment in patients with moderate-to-severe asthma as supporting data is from trials conducted with the mist inhaler.

Short-acting beta$_2$ agonists

Short-acting beta$_2$ agonists (SABAs) (salbutamol) are used as needed for symptom relief. They are referred to as 'relievers'; the term 'rescue medication' may be used in the literature.

Beta$_2$ agonists (long-acting or short-acting) should not be used regularly as monotherapy because patients are at high risk of flare-up due to uncontrolled airway inflammation, and increased risk of asthma-related death despite often having few symptoms.

Use:

salbutamol 100 micrograms per puff (actuation), 1 to 2 puffs by inhalation via MDI, as needed.

Most patients are able to use a pressurised metered dose inhaler (MDI) plus spacer. Rarely, nebulised SABA may be required. If nebulised treatment is necessary, use:

salbutamol 5 mg by inhalation via nebuliser.

Rate of response to treatment

Different clinical features of asthma respond to inhaled corticosteroid (ICS) treatment at different rates, with night-time waking improving rapidly and reliever use improving more slowly.

Night-time symptoms improve rapidly with high-dose ICS, with 50% improvement within a few days and nearly complete disappearance after 1 month; most patients do not need oral corticosteroids to control significant initial symptoms.

Table 14: Rate of response of different measures of asthma control to inhaled corticosteroid

Measure of asthma control	Average time to maximum improvement
night-time waking	1 week
FEV_1	2 months
morning PEF	3 months
reliever use	6 months
airway hyperresponsiveness	18 months

FEV_1 = forced expiratory volume in 1 second; PEF = peak expiratory flow

Based on data using high-dose inhaled corticosteroids; Reddel HK, Jenkins CR, Marks GB, Ware SI, Xuan W, Salome CM, et al. Optimal asthma control, starting with high doses of inhaled budesonide. Eur Respir J 2000;16(2):226-35. <www.ncbi.nlm.nih.gov/pubmed/10968496>

Combination inhaler therapy

Overview

For patients who remain symptomatic despite good adherence and correct technique with inhaled corticosteroids (ICS) alone (Step 2 treatment) increase treatment to

Step 3. Switch to a fixed-dose combination inhaler containing low doses of ICS + LABA as maintenance and reliever therapy.

LABAs (long-acting beta$_2$ agonists) should not be used without ICS (inhaled corticosteroids).

Fixed-dose combination inhalers are more convenient for patients than separate inhalers and, importantly, ensure that the LABA is always accompanied by an ICS. Use of LABA alone is not recommended—patients are at high risk of flare-up due to uncontrolled airway inflammation, and an increased risk of asthma-related death despite often having few symptoms.

Each fixed-dose combination product is available with different ICS doses to allow for 'stepping up', or 'stepping down' once good control has been achieved. Various delivery devices are available, however, these products are currently not available on the Fiji EML.

Modifiable risk factors

Smoking

For patients with asthma, tobacco smoking results in:

- increased asthma symptoms
- increased risk of flare-ups
- reduced efficacy of inhaled corticosteroids—higher doses may be needed
- faster than normal decline in lung function.

Smoking during pregnancy may cause abnormal fetal lung development and is a known risk factor for the baby developing asthma. Exposure to household tobacco smoke places an infant at increased risk of developing asthma.

Ask about current smoking behaviour and provide access to smoking cessation strategies at every opportunity.

Triggers

Triggers for asthma vary widely and differ between patients. Multiple triggers may be involved concurrently for a patient, so take a detailed history. Patients with poorly controlled asthma are more susceptible to asthma triggers. Possible triggers for asthma are listed below.

Food allergies rarely trigger acute asthma; however, a confirmed food allergy is a risk factor for asthma-related death. No individual food item triggers asthma in all people. Intolerance to food chemicals (ie dose-related, non–IgE-mediated reactions) may affect asthma in some patients, but this appears to be rare. Specialist investigation is usually necessary for accurate assessment and diagnosis.

Table 15: Triggers for flare-ups (exacerbations) of existing asthma

[Note 1]

Action	Trigger
Always avoid	cigarette smoke
Avoid or minimise if possible	allergens (eg pollen, dust mite, animal dander)
	airborne or environmental irritants (eg cold or dry air, occupational irritants [Note 2], pollution, smoke)
	drugs associated with asthma exacerbations (eg NSAIDs for patients with aspirin-exacerbated respiratory disease, beta blockers [Note 3])
	dietary triggers (either temperature related [eg cold drinks] or allergy related [for patients with food allergies]) [Note 4]
Manage	respiratory tract infections (consider influenza vaccination)
	comorbidities (eg allergic rhinitis, gastro-oesophageal reflux, nasal polyposis, obesity, inducible laryngeal obstruction)
	physiological and psychological changes (extreme emotions, hormonal changes, pregnancy, sexual activity)

NSAID = nonsteroidal anti-inflammatory drug

Note 1: No individual item triggers asthma in all people.

Note 2: Occupational asthma is most commonly reported where people are working with grains or flour and isocyanates (found in paints) but irritants also include wood dust, cleaning products and other chemicals.

Note 3: If a patient with asthma develops an indication for beta-blocker therapy (eg heart failure, myocardial infarction), start beta-blocker therapy at a low dose under supervision.

Note 4: Food allergies rarely trigger acute asthma; however, a confirmed food allergy is a risk factor for asthma-related death.

Lifestyle factors

Diet

Encourage healthy eating for all patients with asthma. Dietary supplements have not been shown to be of benefit for asthma. Dietary restrictions such as low-salt diets, or avoiding dairy foods or food additives are not recommended without dietetic or medical supervision.

Exercise

Reassure patients that asthma is not a contraindication to physical exercise. Recommend regular physical exercise for both adults and children for its beneficial effect on cardiopulmonary fitness and quality of life.

Symptoms such as breathlessness triggered by exercise may be due to lack of fitness rather than exercise-induced bronchoconstriction. However, breathlessness that worsens in the minutes after stopping exercise is likely to be due to asthma. Exercise-induced bronchoconstriction can be managed effectively and is not a reason to avoid physical exercise.

Comorbid conditions

Overview

Several comorbid conditions can worsen asthma symptom control, risk or management see figure below. Managing these conditions may improve symptom control and should be considered before stepping up asthma treatment. This section provides advice about obesity, gastro-oesophageal reflux disease, rhinosinusitis, and upper airway dysfunction in the context of coexisting asthma.

Obesity

Obesity (defined as a body mass index [BMI] more than 30 kg/m2) is associated with an increased prevalence of asthma. In addition, obese people with asthma report more dyspnoea and asthma-like symptoms than nonobese people; therefore, it is important to confirm the diagnosis of asthma by documenting variable airflow limitation. Obese patients are more likely to have obstructive sleep apnoea and gastro-oesophageal reflux, which can also worsen asthma symptom control.

Weight loss of at least 5 to 10% may result in clinically significant improvements in asthma symptom control.

Gastro-oesophageal reflux disease

The relationship between asthma and gastro-oesophageal reflux disease (GORD) is complex. Patients with asthma have higher rates of GORD based on symptoms or results of 24-hour pH monitoring. It is considered that the presence of GORD may worsen asthma symptom control or contribute to symptoms of coughing. Asthma drugs may cause relaxation of the lower oesophageal sphincter.

Symptomatic GORD should be treated; however, this will not necessarily improve asthma symptoms.

Rhinosinusitis

Rhinitis or chronic rhinosinusitus and asthma commonly coexist. Actively assess for and manage concurrent conditions.

Upper airway dysfunction

Upper airway dysfunction (vocal cord dysfunction) often mimics, and is misdiagnosed as, asthma. Variable upper airway obstruction results from abnormal and intermittent

adduction of the vocal cords or supraglottic muscles during respiration. These episodes can be frightening to the patient. Symptoms do not respond to short-acting $beta_2$ agonists.

Upper airway dysfunction may coexist with asthma, and this can worsen symptoms and lead to overtreatment of asthma if it is not diagnosed and managed appropriately. If upper airway dysfunction is suspected, seek specialist advice.

Nondrug interventions

Breathing exercises

Various breathing exercises including the Butekyo breathing technique have been shown to improve asthma symptoms in the short term but have not been shown to improve lung function or reduce bronchial hyperresponsiveness. The design of many studies of breathing exercises is problematic, and much of the benefit may be due to the relaxation strategies that are often incorporated with breathing techniques. These exercises should be used in conjunction with prescribed maintenance asthma therapy; dosage of existing therapy should not be reduced or stopped.

Complementary medicine

Although there is no good evidence that complementary medicines are beneficial for asthma, patients frequently use them. It is important to facilitate an open discussion and to know which complementary medicines patients are taking because they may interact with prescribed medication. Provide information about the possible harms and benefits of complementary medicine. Ensure patients trialling complementary medicines continue with their usual medications.

Education and skills training

Information

All patients require some core information about asthma and treatment, but the education must be personalised to their asthma severity and their health literacy.

Key information includes:

- an explanation of asthma
- rationale for treatment, and differences between relievers and preventers
- potential adverse effects of treatment
- how to recognise worsening asthma and what to do.

Information alone does not appear to improve health outcomes in adults and adolescents with asthma, although symptoms may improve.

Inhaler technique

Correct inhaler technique (Refer to sample Asthma Action Plan in Appendix) is essential for good asthma outcomes. Up to 90% of patients have incorrect inhaler technique, but most are not aware that they have a problem. Correcting inhaler technique can lead to significant improvement in asthma symptom control.

Up to 90% of patients use their inhaler devices incorrectly.

Before prescribing an inhaler device, train the patient in its use and confirm that they are able to use it correctly. Check inhaler technique at every opportunity by watching the patient using their inhaler, and comparing with a device-specific checklist.

Adherence

If asthma symptom control is poor or flare-ups continue to occur despite apparently adequate treatment, consider poor inhaler technique and adherence. Adherence (compliance) often falls with improvement in symptoms.

Assess adherence using an empathic approach that acknowledges adherence is often incomplete. For example, ask the patient if they find it easier to remember their inhaler in the morning or the evening, or ask them if they use their preventer inhaler less or more than 3 days per week.

Strategies that may improve adherence include:

- using an open, nonjudgmental approach when discussing adherence
- allowing the patient to express their concerns about drugs and devices (including about adverse effects), and addressing these concerns
- improving the patient's understanding of asthma management over time; comprehensive information can rarely be retained after one visit
- explaining the goals of treatment and aiming for concordance with the patient's goals
- keeping treatment simple (eg using once- or twice-daily dosing where possible, using as few devices as possible)
- identifying useful daily associations or medication reminders to improve drug adherence (eg using preventive therapy before meals, or in the bathroom before brushing teeth, or setting reminders on phones or other electronic devices)
- enlisting support of the patient's family and peers
- keeping in touch and using reminder letters or telephone calls.

Guided self-management education

Overview

Guided self-management education that includes a written asthma action plan, self-

monitoring and regular clinical review, results in significant reduction in emergency healthcare use and asthma morbidity.

Conduct a structured program over time to teach skills for detecting and managing deteriorating asthma, and for optimal use of drugs. Important components are:

- information about asthma (written, verbal, visual and/or audio)
- self-monitoring of symptoms and/or peak expiratory flow (PEF) if available
- training in optimal inhaler technique
- provision of an individualised written asthma action plan
- regular medical review

Self-monitoring

Teach patients to monitor their asthma symptoms and to take action if symptoms worsen.

Long-term PEF monitoring is needed by only a small minority of patients with asthma, including those with a history of sudden severe flare-ups, with poor perception of airflow limitation, or with severe asthma. For patients carrying out PEF monitoring, it is easier to see changes from a standardised PEF chart compared to a PEF diary. Patients should consistently use the same chart format.

Written asthma action plan

All patients should have an individualised written asthma action plan that outlines how and when to:

- recognise symptoms of asthma deterioration
- start or change reliever and preventer treatment
- seek medical attention.

Written asthma action plans may be based on asthma symptoms or PEF measurements or both (Refer to sample Asthma Action Plan in Appendix). Plans should be simple, individualised and based on two to four action points. While symptom-based action plans are suitable for the majority of patients, an action plan based on PEF measurements is essential in the case of poor perceivers, overperceivers and patients prone to sudden severe flare-ups. Plans based on PEF measurements should use personal-best PEF rather than predicted PEF for action points.

The principles of written asthma action plans for worsening asthma are outlined below. All patients with asthma must know how and when to obtain prompt medical assistance.

All patients with asthma must know how and when to obtain prompt medical assistance.

There is clear evidence of increased inflammation during flare-ups. Therefore, it is inappropriate to recommend bronchodilator treatment alone until the flare-up is severe enough to require oral corticosteroids.

In the past, guidelines recommended no increase in preventer treatment when asthma worsened; this was on the basis of three placebo-controlled trials that failed to show any benefit from doubling inhaled corticosteroid (ICS) dose. However, in those studies complex criteria were used to identify the onset of a flare-up, patients were required to attend on multiple occasions, adherence was strongly emphasised, and the extra medication was not started until approximately 5 days after asthma had started to worsen.

For patients prescribed conventional maintenance preventer treatment, there is some clinical trial evidence in adults to support quadrupling the dose of ICS in the management of flare-ups. Therefore, the current recommendation is to increase ICS to high dose soon after the onset of a flare-up.

Asthma action plans must be individualised.

Asthma action plans must be individualised to the patient's current treatment regimen, their usual asthma symptoms and pattern of reliever use, and their willingness and ability to self-manage worsening asthma.

Box 2: Principles of written asthma action plans

The principles of written asthma action plans for worsening asthma are:

- increase inhaled reliever dose and frequency
- **early** increase in preventer dose
- start prednisolone for a severe flare-up or if already on a high dose of inhaled corticosteroid
- obtain prompt medical attention
- in an emergency:
  - immediately start first aid treatment using high doses of inhaled reliever
  - call an ambulance or transport the patient to the nearest health facility immediately, and advise that the patient is having a severe asthma attack (flare-up)
  - continue to use the reliever until patient recieves medical attention.

Review and referral

Regular review

A formal review process requires the patient to agree to attend appointments on a regular basis. The medical review should include assessment of:

- asthma symptom control by
  - monitoring symptom control including frequency of use of reliever medication
  - reviewing the diagnosis if there is poor response to current level of treatment
  - assessing risk factors for adverse asthma outcomes
  - monitoring spirometry—if available measure lung function (3 to 6 months after preventer treatment starts, then annually thereafter). Normal year-to-year variation in forced expiratory volume in 1 second (FEV_1) is up to 15%
- treatment issues by
  - checking inhaler technique
  - checking adherence to treatment plan
  - reviewing current medications to determine if a trial of stepping up or stepping down treatment is indicated
  - checking patient understanding of their written asthma action plan
- comorbidities

Frequency of review is determined by several factors including the patient's asthma severity. In general, inhaler technique should be checked by a health professional at every opportunity (at least 6-monthly) and the written asthma action plan should be reviewed annually. Additionally, patients should be reviewed within 48 to 72 hours following discharge from an emergency department or ward after a flare-up. Patients should be reviewed within a week following mild flare-ups that have been self-managed or managed in primary care. Opportunistic review is also recommended (eg when patients attend for repeat prescriptions).

More frequent review is recommended for:

- patients with risk factors for adverse outcomes patients with more severe asthma, even if well controlled (ie requiring high-intensity treatment)—review every 3 months
- pregnant women with asthma—review every 4 to 6 weeks.

Effective communication is essential. All health professionals should ensure the patient understands the meaning of the terms used.

Failure to respond to adequate treatment

Overview

Adults and adolescents may not respond to asthma treatment due to incorrect diagnosis and/or a number of other contributing factors including poor inhaler technique, poor adherence and comorbidities. Provided the diagnosis is correct, addressing these contributing factors can vastly improve patients' asthma symptom control and should be considered before stepping up treatment. However, it may not be possible to completely resolve contributing factors and some patients may require a step-up in treatment to maintain good symptom control.

International guidelines for severe asthma define asthma as 'severe' when, to prevent asthma from becoming uncontrolled, either:

- Step 4 treatment has been required for the last year; or
- oral corticosteroids have been required for at least 50% of the last year.

Guidelines also define asthma as severe if asthma *remains* uncontrolled despite either of the above treatments. This also applies to patients in whom treatment of comorbidities remains incomplete.

Well-controlled asthma that worsens on tapering of high-dose ICS or systemic corticosteroid is also considered severe.

'Uncontrolled' asthma is defined as at least one of:

- poor symptom control
- frequent flare-ups—2 or more flare-ups requiring courses of oral corticosteroids (each course lasting 3 days or more) in the last 12 months
- severe or life-threatening flare-up(s)—at least one flare-up requiring hospitalisation, intensive care unit admission or mechanical ventilation in the last 12 months
- airflow limitation—post-bronchodilator forced expiratory volume in 1 second (FEV_1) less than 80% predicted post-bronchodilator; consider coexisting chronic obstructive pulmonary disease in these patients

Refer patients with severe asthma for specialist assessment and management.

Referral

Specialist consultation is recommended when there has been:

- severe asthma as defined above
- asthma in association with anaphylaxis
- a life-threatening acute asthma flare-up requiring hospital admission and invasive ventilation

- poor self-management
- doubt about the diagnosis of asthma
- suspected occupational asthma
- suspected allergic bronchopulmonary aspergillosis
- a concurrent condition complicating asthma management
- frequent emergency department or urgent general practitioner visits for asthma.

Special categories

Exercise-induced bronchoconstriction

Exercise-induced bronchoconstriction, commonly referred to as exercise-induced asthma, is bronchoconstriction triggered by physical activity in people who already have a diagnosis of asthma. It may be the only, or predominant, symptom of asthma. It is different from the bronchoconstriction that is induced by extreme physical exertion in elite athletes who do not have classical asthma. Regular physical exercise is recommended for both adults and children as part of asthma management for its beneficial effect on cardiopulmonary fitness and quality of life. The amount of physical activity required to trigger asthma symptoms depends on the level of fitness of the person. Ambient conditions can also influence the likelihood of exercise-induced bronchoconstriction (more likely under cold and dry conditions). Symptoms such as breathlessness triggered by exercise are not necessarily due to exercise-induced bronchoconstriction, but may be due to lack of fitness or other causes.

Regular ICS (inhaled corticosteroid) is the most effective strategy for preventing exercise-induced bronchoconstriction.

There are several strategies for preventing bronchoconstriction triggered by exercise. Regular inhaled corticosteroid (ICS) therapy is the most effective. An adequate warm-up (short sprints separated by short breaks) may help. If these measures are inadequate, a short-acting beta$_2$ agonist (SABA), montelukast or a cromone may prevent exercise-induced bronchoconstriction if used before the activity. For patients with persisting exercise-induced bronchoconstriction, despite good adherence and good inhaler technique with ICS, use:

> salbutamol 100 micrograms per puff (actuation), 1 to 4 puffs by inhalation via MDI, 15 minutes before exercise

Titrate dose to achieve optimal effect.

Alternative pre-exercise therapies include cromoglycate, nedocromil and montelukast. Treatment with a SABA together with cromoglycate, nedocromil or montelukast may also be used.

Long-acting beta$_2$ agonists are not recommended for the prevention of exercise-induced bronchoconstriction.

Asthma and pregnancy

Fertility is not reduced in asthma. Asthma is the most common illness to complicate pregnancy. Asthma improves in about one-third of pregnancies and is the same or worsens in two-thirds.

Advise women that uncontrolled asthma is a greater risk to their baby than using asthma medications.

Many women stop asthma treatment when they become pregnant because of their concerns about perceived risks of medications. However, evidence shows that the risk to their baby of uncontrolled maternal asthma is much greater than the risk of taking asthma medications. Ninety-seven per cent to 99% of babies born to women with asthma have no congenital birth defects. Babies born to women with asthma have a slightly greater risk of minor congenital abnormalities compared with those born to women without asthma, but this risk is not influenced by asthma medication.

If a switch of regular preventer therapy is considered, it should be undertaken in the pre-conception period and be safe in pregnancy (Refer to Respiratory Therapy in Pregnancy and Breastfeeding). Once a woman with asthma is pregnant, maintain her current treatment as long as her asthma is well controlled.

Review asthma every 4 to 6 weeks during pregnancy because of the increased risk of flare-ups and the attendant risk to the baby. Treat asthma flare-ups in pregnant women the same as flare-ups in nonpregnant women, ie oral corticosteroids should not be avoided. Strongly advise women with asthma who are pregnant not to smoke, and to avoid exposure to tobacco smoke. Update their asthma management plan and recommend appropriate vaccination.

Asthma in adolescence

Adolescence is generally defined as 12 to 18 years of age. About one-third of adolescents will experience asthma remission during puberty; the reason for this is unknown. However, asthma may improve or worsen in adolescence, or it may be the time of first presentation. For females, flare-ups may be associated with their menstrual cycle.

Give adolescents the opportunity to be seen without parents/carers present. This allows discussion of sensitive issues impacting on asthma management such as tobacco and drug use, adherence and their concerns about their asthma. Assure them appropriate confidentiality will be maintained.

Assess health literacy and provide the appropriate level of verbal and written information.

Be aware of the increased prevalence of risk-taking behaviour in this age group.

Asthma in older people

The prevalence of asthma in this group is the same as in the general adult population. Asthma first presenting in this group is often called late-onset asthma.

Asthma may be harder to diagnose in older people due to under-reporting of symptoms that are assumed to be due to ageing and the presence of concurrent conditions (eg chronic obstructive pulmonary disease [COPD], congestive heart failure, deconditioning). The coexistence of increased airflow variability (asthma) and incompletely reversible airflow limitation (COPD) is common in this age group.

Consider the potential for drug interactions and aggravation of pre-existing conditions (eg tachyarrhythmias, diabetes) when prescribing drugs for asthma.

Choose suitable inhaler devices and train patients in their use. Poor inhaler technique is common, and may be due to mechanical difficulties (eg osteoarthritis), visual or cognitive impairment, and poor inspiratory flow. If possible, avoid prescribing multiple different inhaler devices.

Occupational asthma

Occupational asthma is common, occurring in up to 20% of people with asthma, and is probably underdiagnosed. Consider occupational asthma in all adult-onset asthma. Asthma may be induced *de novo* by occupational exposure to a number of chemicals or allergens. For patients with pre-existing asthma, asthma symptom control may be worsened by workplace exposures.

Ask patients whether asthma symptoms improve on days away from work or during holidays.

The risk of developing occupational asthma may be reduced by avoiding workplace exposure to known sensitisers, such as isocyanates in spray-painting workshops, laboratory animals, and latex gloves in healthcare facilities.

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis (ABPA) is a condition in which a marked inflammatory reaction to the *Aspergillus fumigatus* mould occurs in the airways. Most patients with ABPA have asthma, and the asthma is often severe, so patients with severe asthma should be considered for the presence of ABPA. It may require systemic corticosteroid therapy and/or antifungal treatment.

If ABPA is suspected, refer to a specialist for diagnosis and management.

Aspirin-exacerbated respiratory disease

Aspirin-exacerbated respiratory disease is a specific asthma phenotype, which includes:

- asthma
- nasal polyposis
- aspirin sensitivity (there may be coexisting nonsteroidal anti-inflammatory drug [NSAID] sensitivity)
- chronic hyperplastic eosinophilic sinusitis.

Aspirin-exacerbated respiratory disease often starts with nasal symptoms. Asthma in this group is more likely to be severe, and the onset is usually when patients are in their 20s or 30s. It is more common in females.

Specialist assessment is appropriate. Aspirin challenges are dangerous; desensitisation must only be done under specialist supervision.

Management of acute asthma in adults and adolescents

This topic provides advice for managing acute asthma in adults and adolescents over 15 years. In the event of an acute flare-up (exacerbation) of asthma, early intervention is the best strategy to prevent further deterioration.

Educate all patients with asthma to recognise early symptoms of deterioration and to initiate the first steps of treatment Ongoing medical management is based on a rapid preliminary assessment with initiation of first-line treatment according to severity. This is followed by a more detailed assessment to guide ongoing management. The appropriate disposition (ie care environment) must be anticipated and organised as appropriate.

Suspected anaphylaxis

Anaphylaxis is an important differential diagnosis for patients presenting with acute wheeze. Anaphylaxis is a life-threatening condition; the patient can deteriorate exceedingly rapidly (ie within minutes). This diagnosis is likely if an apparent asthma flare-up occurs with angioedema or rapid onset of a widespread rash. Also consider anaphylaxis if there are marked abdominal symptoms or hypotension.

Intramuscular adrenaline is first-line treatment in suspected anaphylaxis.

Manage anaphylaxis according to appropriate national guidelines or local clinical protocols. If anaphylaxis is suspected or cannot be excluded, intramuscular adrenaline should be given as a priority.

First aid

If symptoms are **severe or life-threatening**, urgently call an ambulance and advise that the patient is having a 'severe asthma attack' (flare-up) and/or organise transport to hospital. Pending hospital arrival, administer high doses of inhaled short-acting beta$_2$ agonist (SABA) via pressurised metered dose inhaler (MDI) with spacer (if available) or via nebuliser. Use:

> salbutamol 100 micrograms per puff (actuation), 6 to 12 separate puffs by inhalation via MDI with spacer, repeated every 20 minutes if indicated, or sooner if required
>
> OR
>
> salbutamol 5 mg by inhalation via nebuliser, repeated every 20 minutes if indicated, or sooner if required.

If early signs of **asthma deterioration** are present, first-line treatment of an acute asthma flare-up should be initiated by the patient or carer according to the patient's asthma plan. An ambulance should be called if there is not a rapid and significant response to first-line home treatment, or if there are signs of a severe or life-threatening flare-up. Urgent medical review is also required if the patient initially responds but bronchodilator therapy is needed more than 3 to 4 hourly.

Seek urgent hospital care if there are signs of a severe or life-threatening flare-up or if the patient fails to respond rapidly and significantly to first-line treatment.

An alternative '**first aid plan**' for acute asthma, called '4×4×4', recommends salbutamol MDI. Refer to Sample Action Plan; if available, use:

- 4 separate puffs, with spacer if available, one puff at a time
- take 4 breaths from the spacer after each puff
- wait 4 minutes and then give another 4 separate puffs
- if the person still cannot breathe normally, call an ambulance and continue giving 4 separate puffs every 4 minutes until the ambulance arrives.

Medical management

Overview

When the patient arrives at the medical facility, perform a rapid assessment to evaluate the severity of the flare-up (see table below on Initial rapid severity assessment), which determines initial management.

After starting initial therapy, undertake a more detailed review to determine subsequent management.

The aim of management is to stabilise the patient with bronchodilators, administer corticosteroids, if indicated, within the first hour, then attempt to lengthen the interval between salbutamol doses.

Patients with a mild–moderate flare-up may be managed in the primary care setting, with ongoing assessment of severity and response to treatment. If the patient's condition worsens, arrange urgent transfer to an acute care facility.

Ongoing monitoring of the response to therapy is critical; the frequency of reassessment is determined by the severity of the flare-up. If the patient is not responding to treatment at the assessed level of severity, treat according to the next level. Be aware that the patient may appear to respond initially and then relapse. This may be due to bronchoconstriction; however, be alert to the possibility of complications such as pneumothorax. Salbutamol, although relatively safe, is not without adverse effects, including paradoxical worsening of respiratory failure at very high doses.

Ongoing monitoring of treatment response is the key to successful management of acute asthma flare-ups.

Early anticipation of the required level of care is also important. This applies both on initial presentation and if the condition either does not respond as expected or subsequently deteriorates. The need for escalation of level of care is determined by limitations of the current care environment and skill levels of available staff, as well as other factors including ease of access to more advanced assistance. In primary care an early decision must be made about the possible need for transfer to hospital; this may be either due to the initial severity of the flare-up or the likely requirement for ongoing monitoring and/or admission. In the emergency department, early involvement of senior staff is desirable for sicker patients. If retrieval to a higher-level facility is anticipated, initiate discussions sooner rather than later.

Early decision must be made about the possible need for transfer to hospital with higher level of care (eg divisional hospital)

In the post-acute phase of care, ongoing management must also be determined and instituted in an appropriate clinical environment. Before discharge certain criteria must be fulfilled and appropriate follow-up arranged.

Assessment

Initial rapid assessment

Perform an initial rapid assessment immediately on presentation to evaluate the severity of the flare-up (see table below). This risk stratification determines the appropriate initial management.

While clinical features can help identify severity of an acute asthma flare-up, they are not specific (either in isolation or in combination); their absence does not exclude a severe or even life-threatening flare-up.

The degree of wheezing is an unreliable indicator of the severity of an acute asthma flare-up; in severe acute asthma it may be essentially absent (ie 'silent chest'). In these cases, the wheeze becomes apparent only as the airway obstruction is relieved.

Wheezing may be absent in severe acute asthma (termed 'silent chest').

The severity category may also change with time either because more information is available (eg results of pulse oximetry or spirometry) or because the situation itself has altered. The change may be either an increase or decrease in severity, therefore ongoing observation is required.

Table 16: Initial rapid severity assessment of acute asthma in adults and adolescents

If features of more than one severity category are present, record the higher (worse) category as the overall severity level.

Mild–moderate: all of the following	Severe: any of the following	Life-threatening: any of the following
• can walk and can speak whole sentences in one breath • SpO_2 more than 94%	• unable to complete sentences in one breath • increased work of breathing with use of accessory muscles (eg tracheal tug, intercostal or subcostal recession, marked abdominal breathing) • obvious respiratory distress • SpO_2 90 to 94%	• reduced consciousness • collapse • soft or absent breath sounds • poor respiratory effort • exhaustion • cyanosis • SpO_2 less than 90%

cont...

Important additional information:

- The severity category may change when more information is available or over time. Ongoing observation is required
- If oxygen therapy has already been started, it is not essential to stop oxygen to measure oximetry.
- Oxygen saturation levels are a guide only and are not definitive; clinical judgment should be applied.
- While clinical features can help identify severity of an acute asthma flare-up, they are not specific (either in isolation or in combination); their absence does not exclude a severe or life-threatening flare-up. Of note:
  - wheezing may be absent in severe acute asthma (ie 'silent chest')
  - pulsus paradoxus is not a reliable indicator of the severity of acute asthma
  - Life-threatening acute asthma can occur without cyanosis.

SpO_2 = oxygen saturation measured by pulse oximetry

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Secondary severity assessment

Following initial administration of salbutamol, undertake a secondary severity assessment, and adjust management accordingly.

Assessment of response to treatment is an ongoing process; frequency of assessment should be tailored depending on the result and will change as the patient's condition changes. Repeat secondary severity assessment frequently within the first hour (eg after each dose of salbutamol). In more severe cases, remain at the bedside until the patient is stabilised.

Table 17: Secondary severity assessment of acute asthma in adults, adolescents and children 6 years or older

If features of more than one severity level are present, treat according to the higher severity level.

	Mild to moderate: all of the following	**Severe: any of the following**	**Life-threatening: any of the following**
Consciousness	alert	may be the same as mild to moderate and does not determine severity	reduced consciousness (eg drowsy or unconscious)

cont...

Speech	can finish a sentence in one breath	can only speak a few words in one breath	can't speak or can only speak single words because of dyspnoea
Posture	can walk	difficulty lying flat because of dyspnoea sitting hunched forward ('tripoding') tiring	collapsed or exhausted
Breathing	respiratory distress is not severe	increased work of breathing with use of accessory muscles (eg tracheal tug, intercostal or subcostal recession, marked abdominal breathing)	severe respiratory distress or poor respiratory effort
Skin colour	normal	may be the same as mild to moderate and does not determine severity	cyanosis (not always present)
Respiratory rate	less than 25 breaths/min	25 breaths/min or more	bradypnoea (indicates respiratory exhaustion)
Heart rate	adults: less than 110 beats/min children: normal range [Note 1]	adults: 110 beats/min or more children: tachycardia [Note 1]	cardiac arrhythmia or bradycardia (may occur just before respiratory arrest)
Chest auscultation	wheeze or persistent cough (not always present)	may be the same as mild to moderate and does not determine severity	'silent chest' due to reduced air entry

cont...

Pulse oximetry	SpO_2 more than 94%	SpO_2 90 to 94%	SpO_2 less than 90% or clinical cyanosis
Lung function tests (only applicable to adults)	FEV_1 or PEF more than 40% of predicted or personal best	FEV_1 or PEF less than 40% of predicted or personal best	not indicated (not usually able to be performed)
Chest X-ray	not usually required; indicated if pneumonia, atelectasis, pneumothorax or pneumomediastinum is suspected, or if sudden deterioration.		
Asthma history	assess risk factors for asthma-related death, see Table 11		

FEV_1 = forced expiratory volume in 1 second; PEF = peak expiratory flow; SpO_2 = oxygen saturation measured by pulse oximetry

Note 1: Normal values for heart rate and respiratory rate in children vary with age:

Heart rate children 5-12 years 80-120 beats/minute; children 12-18 years 60-100 beats/minute

Respiratory rate children 5-12 years 20-25 breaths/minute; children 12-18 years 15-20 breaths/minute

Source: Samuels M, Wieteska S. (Eds) Advanced paediatric life support: the practical approach. 5th edn. Wiley-Blackwell, Oxford; 2011.

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Box 3: Risk factors for potentially fatal asthma in adults and adolescents

Risk factors for potentially fatal asthma are any of the following:

- previous ventilation or intensive care unit (ICU) admission due to asthma
- hospital admission due to asthma in the last year
- repeated emergency department attendances in the last year
- requiring three or more classes of asthma maintenance medication
- frequent short-acting $beta_2$ agonist use (eg more than one canister used per month)
- poor lung function
- history of brittle asthma (ie sudden and severe flare-ups)
- confirmed food allergy
- current or recent oral corticosteroid use
- rural or remote location.

The above factors often coexist with adverse psychosocial or behavioural factors, such as:

- poor adherence, including with drugs and follow-up visits
- depression and/or other psychiatric illness
- substance misuse, including smoking
- denial of seriousness of condition and need for regular treatment
- social isolation
- learning difficulties
- financial, employment and/or domestic problems.

Sources: British Thoracic Society (BTS), Scottish Intercollegiate Guidelines Network (SIGN). British guideline on the management of asthma: a national clinical guideline (SIGN 141). Edinburgh: SIGN; 2014. <www.sign.ac.uk/guidelines/fulltext/141/index.html>

Goeman DP, Abramson MJ, McCarthy EA, Zubrinich CM, Douglass JA. Asthma mortality in Australia in the 21st century: a case series analysis. BMJ Open 2013;3(5). <www.ncbi.nlm.nih.gov/pubmed/23793664>

Treatment of acute asthma in adults and adolescents

Overview

This section provides a summary of the principles of drug treatment of acute asthma.

The early use of inhaled short-acting $beta_2$ agonist therapy (such as salbutamol) is the cornerstone of successful management of all patients with acute asthma, regardless of severity. Give oral corticosteroids within the first hour of management to all patients with an acute asthma flare-up.

Patients with a life-threatening asthma flare-up usually require supplementa oxygen. Early initiation of ipratropium is recommended.

In severe asthma flare-ups, supplemental oxygen is indicated if oxygen saturation is less than 95%. Early ipratropium should be considered.

Depending on response to initial treatment, intravenous magnesium sulfate may be required in severe and life-threatening acute asthma. Other bronchodilators that may be considered in life-threatening acute asthma are intravenous salbutamol and intravenous aminophylline.

Failure to respond to therapy should prompt reassessment. Review the diagnosis, including consideration of complications such as pneumothorax or adverse effects of drug treatment.

Ventilatory support may need to be considered for patients with life-threatening flare-ups. Intubation and ventilation of these patients is difficult and has significant associated risks. Ideally it should only be undertaken by an experienced critical care practitioner. Noninvasive ventilation may be useful in selected patients, but this is controversial and evidence for its use is inconclusive.

Inhaled bronchodilators

Inhaled salbutamol is indicated for all episodes of acute asthma. Give salbutamol via pressurised metered dose inhaler (MDI) with spacer to those with mild–moderate acute asthma. For patients with severe acute asthma or those unable to use a MDI with spacer, administer salbutamol via intermittent nebulisation. For patients with life-threatening acute asthma, administer salbutamol via continuous nebulisation.

Although relatively safe, salbutamol has a range of adverse effects. Hypokalaemia and hypomagnesaemia are expected when repeated or high doses are given and should be managed appropriately. The effects on the cardiovascular system may lead to adverse consequences such as myocardial ischaemia or prolonged QT interval; the latter predisposes to arrhythmias, especially in the context of electrolyte disturbances.

High doses of salbutamol may paradoxically worsen the respiratory compromise. This effect is thought to be multifactorial; metabolic acidosis, rise in lactate levels and increased metabolic rate may all contribute. If suspected, the patient should be closely observed, and salbutamol treatment should be cautiously back-titrated; specialist input is recommended.

If there is poor response to initial inhaled salbutamol, add inhaled ipratropium. Patients with more severe asthma benefit the most from the addition of ipratropium.

Corticosteroids

Give oral prednisolone within the first hour of management for all patients with an acute asthma flare-up. If the oral route is not possible, intravenous hydrocortisone or dexamethasone (only if hydrocortisone unavailable) can be used.

For oral corticosteroid therapy, use:

1. prednisolone 37.5 to 50 mg orally, within 1 hour of presentation; continue once daily for a total of 5 to 10 days

OR

2. dexamethasone 16 mg orally, within 1 hour of presentation; repeat dose once the next day.

If oral therapy is not tolerated, use:

hydrocortisone 100 mg intravenously, 6-hourly for up to 24 hours; switch to an oral corticosteroid once tolerated. If intravenous therapy is still required after 24 hours, reduce frequency to 12-hourly.

Management of mild to moderate acute asthma

For a patient with mild to moderate acute asthma according to the initial rapid assessment, start treatment with salbutamol. Use:

salbutamol 100 micrograms per puff (actuation) 4 to 12 acutations (1 at a time) with a spacer; repeat as required.

Immediately after starting treatment, perform a secondary severity assessent to determine ongoing treatment.

All adults with moderate acute asthma should also receive **oral corticosteroid** therapy as soon as practical (and at least within the first hour of presentation). See next section for doses.

Management of severe acute asthma

For a patient with severe acute asthma according to the initial rapid assessment, start treatment with both salbutamol and ipratropium.

If oxygen saturation measured by pulse oximetry (SpO_2) is less than 92%, also start supplemental oxygen therapy.

Immediately after starting treatment, perform a more detailed secondary severity assessment to determine ongoing treatment.

All patients should also receive oral corticosteroid (or intravenous if oral intake is not possible) therapy as soon as practical (and at least within the first hour of presentation). See below for doses.

For bronchodilator therapy in a patient with severe acute asthma, use:

> salbutamol 100 micrograms per puff (actuation) 12 acutations (1 at a time) via MDI with a spacer; repeat every 20 minutes for the first hour (or sooner if needed)
>
> PLUS
>
> ipratropium 21 micrograms per puff, 8 puffs (1 at a time) via MDI with spacer, repeat every 20 minutes for the first hour (or sooner if needed)

Bronchodilator therapy can be administered by intermittent nebulisation if the patient can't breathe through a spacer. Mix the nebuliser solutions and drive the nebuliser with air unless oxygen required.

> salbutamol 5 mg via intermittent nebulisation every 20 minutes for the first hour (or sooner if needed)
>
> PLUS
>
> ipratropium 500 micrograms via intermittent nebulisation every 20 minutes for the first hour (or sooner if needed).

If symptoms improve or resolve, bronchodilator treatment can be used less frequently or stopped. Monitor the patient for at least an hour after symptom resolution in case symptoms recur.

If the patient worsens or does not have a rapid and marked response to treatment, consider escalating treatment to that used for life-threatening acute asthma. Intravenous magnesium sulfate may be required. If the patient is being managed in primary care, arrange transfer to a hospital.

Management of life-threatening acute asthma

Consider anaphylaxis as a differential diagnosis—see 'Anaphylaxis and acute asthma' for more information.

For a patient with life-threatening acute asthma according to the initial rapid assessment, arrange immediate transfer to a critical care or high-dependency facility. Early involvement of senior staff is desirable for very sick patients.

Start immediate treatment with nebulised bronchodilator therapy (both salbutamol and ipratropium). Supplemental oxygen therapy is almost always required in these patients—use to drive the nebuliser.

All patients should also receive oral corticosteroid (or intravenous if oral intake is not possible) therapy as soon as practical (and at least within the first hour of presentation). See next section for doses.

Immediately after starting treatment, perform a more detailed secondary severity assessment. Depending on the response to initial treatment, intravenous magnesium sulfate may be required. Additional therapy or ventilatory support may be required in difficult cases.

Continuously reassess severity and response to treatment to determine ongoing management.

If the patient is unresponsive, has poor respiratory effort, and cannot inhale bronchodilators, or is considered to be peri-arrest, consider adrenaline (epinephrine).

Oxygen therapy

If oxygen saturation measured by pulse oximetry (SpO2) is less than 92%, start supplemental oxygen (use oxygen to drive the nebulised bronchodilator therapy). Supplemental oxygen is almost always required for patients with life-threatening acute asthma.

Titrate oxygen to a target SpO2 of 92 to 96%. For more detailed advice on supplemental oxygen administration, see ‘Acute oxygen therapy.’

Bronchodilator therapy

Start immediate treatment with nebulised salbutamol and ipratropium, use oxygen to drive the nebuliser:

> salbutamol 2.5 mg at a time via continuous nebulisation
>
> PLUS
>
> ipratropium 250 micrograms added to nebulised solution every 20 minutes for the first hour.

Hypokalaemia and hypomagnesaemia are likely with repeated high doses of salbutamol—anticipate and manage early. Cardiovascular effects (eg myocardial ischaemia, prolonged QT interval predisposing to arrhythmias) can also occur.

If the patient shows a marked improvement with initial treatment, consider switching delivery of bronchodilator therapy to intermittent nebulisation or MDI with spacer. For doses, see ‘ .’

If severe or life-threatening acute asthma persists after starting treatment, give intravenous magnesium sulfate.

Oxygen

If oxygen saturation measured by pulse oximetry (SpO_2) is less than 95%, give supplemental oxygen and titrate to a target SpO_2 of 92 to 96%.

Intravenous magnesium sulfate

There is strong evidence that magnesium sulfate has bronchodilator effects. The sickest patients appear to be the most likely to benefit from intravenous magnesium sulfate. Guidelines recommend considering intravenous magnesium sulfate in severe and life-threatening acute asthma after first-line bronchodilator therapy (ie salbutamol and ipratropium) has been initiated.

Comprehensive monitoring in a high-dependency–type environment is required.

For severe or life-threatening acute asthma that has not responded to initial treatment, use:

> magnesium sulfate 10 mmol diluted to 100 mL in a compatible fluid, by slow intravenous injection over 20 minutes.

Magnesium sulfate is administered as a single short intravenous infusion; the safety of repeated doses has not been assessed. Hypermagnesaemia may cause loss of deep tendon reflexes and muscle weakness, including respiratory muscle weakness.

Additional treatment for persistent life-threatening acute asthma

Adrenaline (epinephrine)

If the patient is unresponsive, has poor respiratory effort, and cannot inhale bronchodilators, or is considered to be peri-arrest, consider adrenaline (epinephrine). Give:

> adrenaline (epinephrine) 1 mg/mL (1:1000, 0.1%) solution, 0.01 mg/kg up to 0.5 mg (0.5 mL) intramuscularly; repeat after 3 to 5 minutes if required.

In life-threatening situations adrenaline (epinephrine) can also be administered intravenously or by continuous infusion—seek expert advice before administration.

Intravenous salbutamol

The potential harm versus benefit of intravenous salbutamol is not favourable compared to administration via nebulisation; there is no evidence of increased efficacy. Intravenous salbutamol is therefore reserved for the sickest patients and/or when nebulisation is impractical.

Comprehensive monitoring in a critical care environment is required.

Use:

salbutamol 200 to 300 micrograms intravenously over 1 minute, then:

repeat after 15 minutes as required, or

infuse at 5 micrograms/minute; adjust according to response (usual rate 10-20 micrograms/minute)

Intravenous aminophylline

The potential harm versus benefit of intravenous aminophylline is considered unfavourable in the majority of situations. It can cause vomiting, arrhythmias, convulsions and sudden death, and is rarely used. Aminophylline interacts with many medicines, including antibiotics such as erythromycin and ciprofloxacin. Check current medications before starting treatment.

The occasional patient with life-threatening acute asthma who is not improving with initial therapy, may respond favourably to aminophylline.

Comprehensive monitoring in a critical care environment is required.

Use **ideal body weight** to calculate dose for obese patients.

Adults not previously treated with theophylline:

loading dose, 5 mg/kg intravenously, given at a rate of <25 mg/minute

maintenance dose, 0.5 mg/kg/hour intravenously

Dose equivalence: 1 mg aminophylline is equivalent to 0.8 mg theophylline.

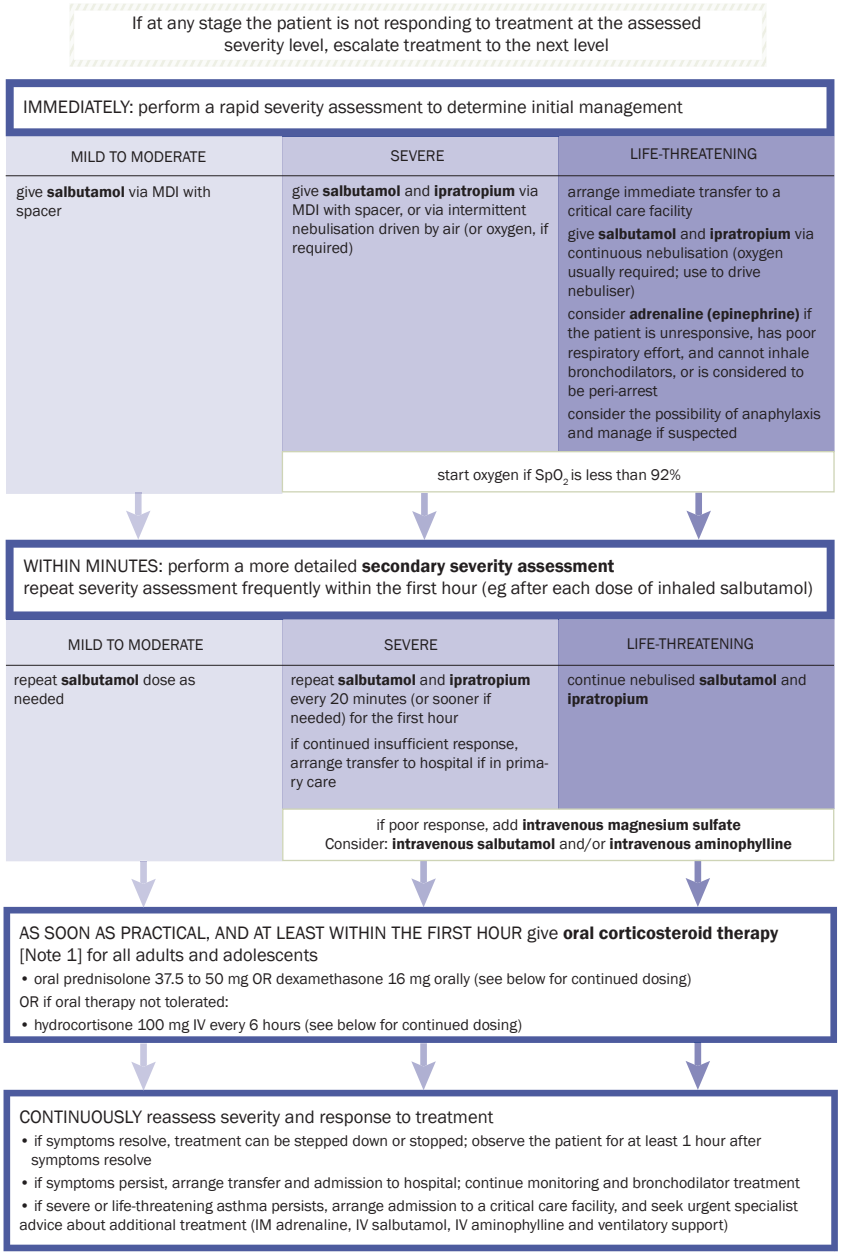
Ventilatory support

Ventilatory support may be required for patients with persistent life-threatening acute asthma.

Consider noninvasive positive pressure ventilation for patients who continue to show no improvement with treatment and are starting to tire or show signs of respiratory failure. Evidence for its use is limited, particularly in children; however, it may avoid the need for intubation. High-flow nasal cannula oxygen therapy is used in some centres, but evidence is limited. Adequately trained staff are needed to administer and monitor acute noninvasive ventilation, usually in a critical care environment or a high-dependency unit; seek expert advice before administration.

If the patient doesn't improve with noninvasive ventilation, intubate and start mechanical ventilation. Intubation and ventilation of these patients is difficult and has significant associated risks. Ideally, it should only be undertaken by an experienced critical care physician.

Figure 3: Summary of management of acute asthma in adults and adolescents



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MDI = metered dose inhaler; Sp02 = oxygen saturation measured by pulse oximetry.
Note 1: Use intravenous corticosteroid therapy if oral intake is not tolerated.
References: Australian Asthma Handbook © 2020. National Asthma Council Australia.
UpToDate

Table 18: Drug dosages for acute asthma in adults and adolescents

Drug [Note 1]	**Delivery**	**Dosage**
salbutamol	MDI with spacer	100 micrograms × 6 to 12 separate puffs
	intermittent nebulisation	5 mg
	continuous nebulisation driven by oxygen [Note 2]	5 mg × 2 nebules
	IV [Note 3]	Loading dose 200 micrograms over 1 minute. Then maintenance infusion of 5 micrograms/minute, adjust according to response (usual rate 10 to 20 micrograms/minute). Use only in critical care units with adequate monitoring.
ipratropium	MDI with spacer	21 micrograms × 8 separate puffs
	intermittent nebulisation	500 micrograms
corticosteroids	oral	prednisolone 37.5 to 50 mg daily; continue for 5 to 10 days
	IV [Note 4]	hydrocortisone 100 mg every 6 hours
oxygen [Note 2]	intranasal	start if Sp02 is less than 95% and titrate to target Sp02 of 92 to 96%
magnesium sulfate [Note 5]	IV infusion	10 mmol diluted in 100mL sodium chloride 0.9%, given over at least 20 minutes, as a single dose Comprehensive monitoring in a critical care or high-dependency environment is required.

cont...

aminophylline [Note 3]	IV loading dose [Note 6], followed by IV infusion	IV loading dose (omit loading dose in patients already taking oral theophylline): 5 mg/kg (up to 500 mg) FOLLOWED BY IV infusion: 0.5 mg/kg/hour Target theophylline plasma concentration: 55 to 110 micromol/L (10 to 20 mg/L); measure daily As no therapeutic drug monitoring available in Fiji, monitor closely for signs of toxicity (eg vomiting). Toxicity can result in seizures and arrhythmias at high concentrations.

IV = intravenous; MDI = pressurised metered dose inhaler; SpO2 = oxygen saturation measured by pulse oximetry

Note 1: Drug, dose, frequency and delivery method depend on severity of acute asthma

Note 2: Continuous nebulisation driven by oxygen is indicated in life-threatening acute asthma. Patients with severe acute asthma using intermittent nebulisation or MDI with spacer may require oxygen; administer oxygen separately.

Note 3: Only indicated in life-threatening acute asthma); comprehensive monitoring is required in a critical care environment.

Note 4: Intravenous corticosteroids are only indicated if the oral route is not available. Change to oral corticosteroids as soon as possible.

Note 5: Only indicated in severe and life-threatening acute asthma; comprehensive monitoring is required in a high-dependency–type environment.

Post-acute care

Patients with any feature of acute life-threatening asthma require formal admission.

Patients with any feature of acute severe asthma persisting after initial treatment also require admission, which may be to a ward or, in some cases, a short stay observation unit.

The extent of ongoing management of acute asthma, including decisions about admission and discharge, is also influenced by the patient's:

- response to therapy
- past history of flare-ups
- current circumstances (eg time of day, distance from medical help, access to phone, home environment)

- treatment adherence
- comorbidities
- risk factors for potentially fatal asthma.

Note that there is no single physiological measurement that can define whether a patient is safe for discharge.

There is no single physiological measurement that can define whether a patient is safe for discharge.

In addition to the above, consider spirometry or peak expiratory flow (PEF) findings (these can often be performed after 1 hour of management); a low forced expiratory volume in 1 second (FEV1) (eg less than 60% predicted) or PEF may indicate a need for hospital admission.

Patients with severe asthma and one or more adverse psychosocial factors are at risk of asthma-related death (see Box 3).

Patients with severe asthma and one or more adverse psychosocial factors are at risk of asthma-related death.

Before discharge, ensure the patient's medication can be continued at home (eg switch nebulised bronchodilators to MDI with spacer). Only discharge the patient if they require short-acting $beta_2$ agonist (SABA) less frequently than every 4 hours; prescribe as-needed SABA on discharge. Continue oral corticosteroids for 5 to 10 days, including the doses administered in the acute phase of management.

Any patient with a flare-up severe enough to require treatment at a health facility, and who is not already using regular inhaled corticosteroid (ICS) treatment, should be started on regular ICS treatment to reduce the risk of another flare-up.

Any patient with a flare-up severe enough to require treatment at a health facility should be started on regular ICS treatment.

A number of important reviews also need to be completed before discharge; see Box 4.

Box 4: Reviews required before discharging an adult or adolescent after an acute asthma flare-up

Following the resolution of an acute asthma flare-up, review:

- inhaler technique
- adherence to prescribed drug regimen
- triggers to identify the possible cause of this flare-up; discuss avoidance measures if necessary
- inhaled corticosteroid dose; adjust the patient's maintenance therapy if necessary
- written asthma action plan including documented advice about escalating treatment and when to seek medical assistance; educate the patient about the action plan.

Follow-up

It is essential that a patient who suffers an acute asthma flare-up, of any severity, be followed up. The patient should be medically reviewed, preferably within 48 to 72 hours of discharge from the emergency department, ward or health facility. Provide a discharge summary in time for this review, including details of the:

- severity of the flare-up
- spirometry, if available
- treatment administered
- treatment prescribed at discharge
- written asthma action plan provided to the patient.

Patients with near-fatal or brittle asthma (ie sudden and severe flare-ups) should have ongoing specialist review for life, while those with a severe flare-up should receive specialist review for at least 1 year. Consider specialist referral for any first presentations of asthma in adults and adolescents and for all asthma presentations requiring admission. Also consider referral to a specialist for patients with ongoing poorly controlled asthma.

Rhinitis and rhinosinusitis

Rhinitis

Introduction

Rhinitis is inflammation of the mucosal lining of the nose causing symptoms including anterior and posterior rhinorrhoea, sneezing, nasal blockage, itching of the nose and loss of the sense of smell. The commonest causes are infections and allergy.

Rhinitis and asthma commonly coexist, a concept sometimes referred to as 'United Airway Disease'. Asthma occurs in 30% of patients with allergic rhinitis, and allergic rhinitis occurs in more than 80% of patients with allergic asthma. Chronic rhinosinusitis also occurs commonly in these patients. Always assess and manage both upper and lower airway disease.

While most rhinitis is allergic, other categories of rhinitis exist including nonallergic rhinopathy (previously called vasomotor rhinitis), infectious rhinitis, occupational rhinitis and drug-induced rhinitis.

Allergic rhinitis

Clinical features and classification

Allergic rhinitis is the most common form of noninfectious rhinitis and is associated with an immunoglobulin E (IgE)-mediated immune response. It is often associated with ocular symptoms and sinusitis. The prevalence of allergic diseases, including allergic rhinitis, has increased in recent decades; they now affect 10 to 30% of the world's population.

Allergic rhinitis is classified in terms of symptom duration (intermittent or persistent) and severity (mild or moderate-to-severe). It was previously defined as seasonal allergic rhinitis (hay fever) or perennial allergic rhinitis, according to the different allergic triggers implicated: seasonal allergens such as grass, weed and tree pollens or perennial allergens such as dust mites, cat and dog dander. However, most patients are sensitised to both seasonal and perennial allergens.

Allergic rhinitis and asthma often coexist; always assess and manage both upper and lower airway disease. Atopic dermatitis (eczema) is also commonly associated.

Allergic rhinitis is caused by environmental allergens (seasonal or perennial) and can be aggravated by chemical irritants (eg active or second-hand smoking). If there is a clinically obvious allergen or irritant, it may be possible to minimise exposure. Check also for possible overuse of intranasal decongestants (rhinitis medicamentosa).

Table 19: Classification of allergic rhinitis by symptom duration and severity

Duration of symptoms	
Intermittent	*Persistent*
symptoms present: on less than 4 days a week OR for less than 4 consecutive weeks	symptoms present: on more than 4 days a week AND for more than 4 consecutive weeks
Severity of symptoms	
Mild	*Moderate to severe*
all of the following present: • symptoms present but not troublesome • normal sleep • no impairment of daily activities, leisure and/or sport • no impairment of school or work performance	one or more of the following present: • troublesome symptoms • sleep disturbance • impairment of daily activities, leisure and/or sport • impairment of school or work performance

Drug treatment

Overview

The drug treatment of allergic rhinitis is outlined below according to symptom duration and severity. Drug doses are provided in the following text. Intranasal saline (sodium chloride solution) may be useful as adjunctive therapy with any of the treatments.

In intermittent, or mild-to-moderate allergic rhinitis, drugs of different classes may be used in sequence, or in combination from the start, according to the age of the patient, degree of disability and cost of therapy. Generally, intranasal or oral antihistamines are used for mild, intermittent symptoms and oral antihistamines are especially useful for associated ocular symptoms. Antihistamines are less effective for nasal obstruction, whereas intranasal corticosteroids are more helpful for this symptom and for more severe disease. Antihistamines have a fast onset of action making them a useful rescue medication; in contrast, intranasal corticosteroids have a slow onset of action.

Undertake a full trial of drug treatment of at least 1 month and verify adherence. Adherence may be poor due to cost of drug treatment or because the patient stops or reduces treatment when their symptoms improve. If there is no response to treatment reconsider the patient's diagnosis.

Features such as unilateral symptoms, nasal obstruction without other symptoms, pain, purulent discharge, recurrent epistaxis and anosmia suggest an alternative diagnosis. In these circumstances and while treatment is continued, a computed tomography (CT) scan of the sinuses and/or referral to the ENT Clinic is appropriate. A CT scan delivers significant local radiation; for sinus CT this includes to the lens of the eye, which is associated with an increased risk of cataracts.

Table 20: Treatment of allergic rhinitis

Symptoms			
Intermittent		**Persistent**	
Mild	**Moderate to severe**	**Mild**	**Moderate to severe**
oral or intranasal antihistamine	oral or intranasal antihistamine *other options are:* intranasal corticosteroid OR montelukast [Note 2]	oral or intranasal antihistamine AND/OR EITHER intranasal corticosteroid OR montelukast [Note 2]	intranasal corticosteroid AND EITHER oral or intranasal antihistamine [Note 1] AND/OR montelukast [Note 2]
	Review after 2 to 4 weeks *If symptoms improve:*		
	• continue for additional 1 month	• continue for additional 1 month	• treat as for persistent mild symptoms and continue treatment for more than 1 month
	If no improvement:		

cont...

	• review diagnosis and adherence • increase intranasal corticosteroid dose and/or determine best combination therapy • consider referral for immunotherapy [Note 3]. Refer to specialist if above steps fail.	• review diagnosis and adherence • treat as for persistent moderate to severe symptoms	• review diagnosis and adherence and possible other causes (eg infection, intranasal decongestant use) • increase intranasal corticosteroid dose and/or use all 3 therapies • If rhinorrhea present, add intranasal ipratropium. • Consider referral for immunotherapy [Note 3]. Refer to specialist if above steps fail.
In addition to the above treatment regimen: Intranasal saline as an adjunctive therapy may be useful Allergen and/or irritant avoidance may be possible see 'Treatment of allergic conjunctivitis' if conjunctivitis is an associated problem. Note 1: An intranasal antihistamine and corticosteroid combination spray is available. Note 2: In children with asthma and allergic rhinitis, montelukast may be selected ahead of an intranasal corticosteroid as it may be effective in both conditions; administration of intranasal corticosteroids can be difficult in children with allergic rhinitis. Note 3: Immunotherapy was not available in Fiji at the time of printing.			

Oral antihistamines

For rapid relief of symptoms of allergic rhinitis such as sneezing and rhinorrhoea, an oral antihistamine can assist and ease both nasal and ocular symptoms. Use:

promethazine 20 to 50 mg orally (child 2 to 12 years 0.5mg/kg up to 50 mg) once daily at night.

OR

loratadine 10 mg (child 1 to 2 years: 2.5 mg; child 2 to 12 years and less than 30 kg: 5 mg; child 2 to 12 years and more than 30 kg: 10 mg) orally, once daily in the morning non-EML

Loratadine and other non-sedating antihistamines are available in the private sector but there is no non-sedating antihistamine on the Fiji EML at the time of publishing.

Intranasal corticosteroids

Intranasal corticosteroids are particularly useful for more severe allergic rhinitis. They are more effective than oral antihistamines and are especially effective for congestive symptoms. Systematic analyses also indicate significant reduction of ocular symptoms.

It is important to explain to patients that intranasal corticosteroids do not relieve symptoms at the time of use; their role is to prevent symptoms. They usually start relieving symptoms within a few days but a minimum trial of a month is needed to establish efficacy.

For both symptom relief and prevention with intranasal corticosteroids, the initial dose is used for 1 month then reduced to a maintenance dose. Use:

Adults:

beclometasone 50 micrograms/spray **initial dose**: 2 sprays into each nostril twice daily for one month, then reduce to **maintenance dose**: 1 spray into each nostril twice daily

Children 6 years and older:

beclometasone 50 micrograms/spray **initial dose**: 1-2 sprays into each nostril twice daily for 1 month, then reduce to **maintenance dose**: 1 spray into each nostril twice daily

To reduce the likelihood of systemic adverse effects, use the minimum dose needed to control symptoms. Tailor the duration of treatment to the patient's symptoms and any drug adverse effects. Intranasal corticosteroid treatment may need to be continued for lengthy periods, even for many years.

Instruct patients on how to administer nasal sprays correctly. Using a crossover technique (right hand to left nostril, left hand to right nostril) reduces the deposition of a corticosteroid directly onto the nasal septum, with less likelihood of causing nasal septal perforation.

Decongestants

Intranasal decongestants (eg oxymetazoline, tramazoline, xylometazoline) produce vasoconstriction in the nasal mucosa, decreasing nasal blood flow and congestion. Consult the product information for directions on use. Intranasal decongestants

should not be used for more than 5 days because prolonged use causes rebound congestion (rhinitis medicamentosa), which can be difficult to manage.

Intranasal or oral decongestants may be useful as short-term therapy in some cases of acute rhinosinusitis or when flying. Decongestants are not recommended for use in children younger than 6 years because significant adverse effects have been reported after use of both nasal and oral decongestants in this age group. Caution should be used in children aged 6 to 12 years. Some paediatric oral formulations containing a decongestant also contain a sedating antihistamine, and there are additional safety concerns for use of these combinations in children.

These products are not on the Fiji EML but are available without prescription from community pharmacies.

Treatment of allergic conjunctivitis

Allergic conjunctivitis can be the predominant symptom in some patients with allergic rhinitis (allergic rhinoconjunctivitis). Oral antihistamines, intranasal corticosteroids and saline (sodium chloride solution) eye drops may be adequate to reduce ocular symptoms.

Use of eye drops containing preservatives is generally not recommended while wearing soft contact lenses. Patients should seek advice if wearing contact lenses and needing to use any eye drops.

Nonallergic rhinitis

Nonallergic rhinitis that is not due to infection includes:

- occupational rhinitis, in response to physical or chemical irritants
- drug-induced rhinitis
- rhinitis triggered by hormonal changes, including during pregnancy
- atrophic rhinitis.

Drugs can cause rhinitis by different pathological or pharmacological mechanisms. Aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), vasodilator drugs (eg some antihypertensives), intranasal decongestants (causing rhinitis medicamentosa) and oral contraceptives can all cause drug-induced rhinitis.

Management of nonallergic rhinitis involves minimising irritant exposure and stopping provocative drugs.

Rhinosinusitis

Introduction

Rhinitis and sinusitis usually coexist, and inflammation of the nasal mucosa and paranasal sinuses is concurrent in most patients. Rhinosinusitis is classified as acute or chronic (symptoms lasting longer than 12 weeks) and is usually due to a viral infection.

Acute rhinosinusitis

Uncomplicated acute viral rhinosinusitis (common cold), the commonest cause of rhinosinusitis, resolves in 7 to 10 days without specific treatment. Viral and bacterial rhinosinusitis are not easily distinguished clinically—use symptomatic therapy alone in all patients initially and **avoid routine use of antibiotics**.

Chronic rhinosinusitis

General information

Chronic rhinosinusitis is a multifactorial disease, defined by symptoms lasting for longer than 12 weeks. Contributing factors include bacterial infection, allergy, cystic fibrosis, physical obstruction (including nasal polyps or anatomical variation), swelling of the mucosa for any other reason, mucociliary impairment, immune deficiency or prolonged use of intranasal decongestant spray.

The diagnosis of chronic rhinosinusitis is primarily clinical and is characterised by two or more symptoms, including nasal blockage/obstruction/congestion or nasal discharge (anterior or posterior). There may also be:

- facial pain or pressure
- reduced sense of smell and taste
- cough
- nausea.

Patients with coexisting allergic rhinitis may also have symptoms such as sneezing, watery rhinorrhoea, nasal itching and itchy watery eyes.

Sinus X-rays are rarely helpful and computed tomography (CT) scans of the sinuses are only indicated for patients failing medical therapy or those with atypical or severe disease (such as with unilateral symptoms, bloodstained discharge, displacement of the eye or severe pain). A CT scan delivers significant local radiation; for sinus CT this includes to the lens of the eye, which is associated with an increased risk of cataracts. If a CT scan is performed, chronic rhinosinusitis will show mucosal changes in the osteomeatal complex or sinuses. CT scans and endoscopic abnormalities should always be interpreted in the context of clinical symptoms because there is a high false positive rate.

Nasal endoscopy of a patient with chronic rhinosinusitis will have at least one objective finding, such as:

- polyps
- mucopurulent discharge from the middle meatus
- oedema or obstruction at the middle meatus.

There are two subtypes of chronic rhinosinusitis, which require different management approaches. They are:

- chronic rhinosinusitis without nasal polyps, representing about two-thirds of cases
- chronic rhinosinusitis with nasal polyps. A polyp appears macroscopically as a grape-like pearly or greyish-yellow–coloured structure markedly different in colour from the nasal mucosa. It may be easily visible inside the nasal cavity.

Chronic rhinosinusitis without nasal polyps

If allergy is suspected as an aetiological factor of chronic rhinosinusitis without nasal polyps, initial management is as for allergic rhinitis. Additional therapies include isotonic or hypertonic saline nasal irrigation (sodium chloride solution) and mucolytics. If there is inadequate response to at least 1 month of treatment or if there is an obvious physical obstruction, refer for specialist management.

There are no data showing efficacy of oral corticosteroids in chronic rhinosinusitis without nasal polyps; however, international guidelines suggest they may be used as short-term rescue medication for symptoms uncontrolled by saline nasal irrigation, intranasal corticosteroids and short-term antibiotics. In this situation, for adults, continue intranasal corticosteroids and add:

> prednisolone 20 mg orally, once daily in the morning for 5 to 10 days.

There are no placebo-controlled trials evaluating the use of antibiotics in chronic rhinosinusitis. Oral antibiotics may be indicated if pus is seen on nasal endoscopy. The choice is preferably based on culture results as the bacteria involved differ from those in acute rhinosinusitis. In the absence of a culture result, give empirical antibiotic treatment with amoxycillin+clavulanate or cefuroxime. Treat for 10 to 14 days depending on patient response.

For patients who do not respond to 2 weeks of treatment with antibiotics, longer courses (up to a month) have been used; there are no placebo-controlled trials to support this practice. There are inadequate data to recommend longer-term antibiotic treatment, such as macrolides, in the primary care setting.

Surgery should be considered for patients with chronic rhinosinusitis unresponsive to the above measures, repeated acute exacerbations, and/or complications of the condition.

Chronic rhinosinusitis with nasal polyps

In children, the presence of nasal polyps should always prompt testing for cystic fibrosis. In adults, check for coexisting asthma and aspirin sensitivity.

Saline nasal irrigations (sodium chloride solution) and intranasal corticosteroids are first-line treatment. If there is inadequate response to intranasal corticosteroids after at least 1 month of treatment, or if the nose is too blocked to be able to use a nasal spray effectively, add a reducing course of oral corticosteroids ('medical polypectomy'). Use:

> prednisolone 20 mg orally, daily for 1 week, then 10mg daily for 1 week, then 10mg on alternate days for 1 week.

If a course of oral prednisolone is ineffective, or symptoms recur, consider specialist referral.

Surgery is indicated for those who fail medical treatment and remain symptomatic. Recurrence of nasal polyps after surgery is usually less than 20%. It is important to continue intranasal corticosteroid therapy to delay or prevent nasal polyp recurrence postsurgery, and this therapy may be needed long term.

Chronic rhinosinusitis with nasal polyps may have a fungal aetiology resulting in conditions such as allergic fungal sinusitis and invasive fungal sinusitis. The fungal infection may need longer medical treatment and more aggressive surgical approaches. Seek specialist review.

Sensitivity to aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) is associated with chronic rhinosinusitis with nasal polyps as well as with asthma; it is called aspirin-exacerbated respiratory disease Selected patients may benefit from aspirin desensitisation but aspirin challenges are dangerous and must only be done under specialist supervision.

Chronic obstructive pulmonary disease

Diagnosis and assessment

Overview

Chronic obstructive pulmonary disease (COPD) is characterised by persistent airflow limitation due to varying combinations of small airways disease and alveolar destruction. It is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, most commonly tobacco smoke, and is generally progressive.

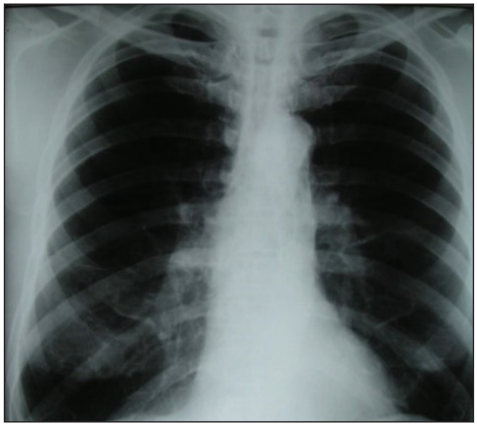
Note that chronic bronchitis, defined as the presence of cough and sputum production for at least 3 months in each of 2 consecutive years, is not necessarily associated with airflow limitation. Emphysema, defined as destruction of the alveoli, is a pathological term often correlated with computed tomography (CT) findings, but it is often not an accurate clinical descriptor.

A chest X-ray can be useful to exclude other causes of breathlessness. A computed tomography (CT) scan is not required for diagnosis of COPD.

It is possible for a patient to have expiratory airflow limitation in the absence of airway obstruction, because airflow limitation may be due to other causes such as loss of tethering or emphysema. Airflow limitation in COPD can be due to loss of supporting elastic recoil from the lung tissue and/or luminal narrowing as a result of airway wall thickening. The term ‘limitation’ is used rather than ‘obstruction’, as obstruction may not always be present (although inherent in the descriptor).

Cigarette smoking is the most important risk factor for COPD; clinically significant COPD is present in around 25% of smokers. However, not all patients with COPD have a history of smoking, especially in countries where exposure to biomass smoke for heating and cooking is common. Other risk factors include maternal smoking, long-standing asthma, respiratory symptoms in childhood, exposure to second-hand smoke, occupational exposures to dusts and fumes, and genetic susceptibility.

Image 2: An example of an X-Ray image in COPD



Initial presentation

Chronic obstructive pulmonary disease (COPD) typically affects middle-aged and older people, with tobacco smoke the major causative factor. Inherited conditions such as alpha1-antitrypsin deficiency render patients more susceptible to the damaging effects of tobacco smoke, leading to early development of COPD.

Consider the possibility of COPD in all patients older than 35 years who are smokers, ex-smokers or have other relevant exposures or predisposing factors, and who present with symptoms including:

- breathlessness
- cough
- recurrent respiratory tract infection
- sputum production
- wheezing.

Breathlessness, which may be the patient's only symptom, typically occurs only on exertion initially, but worsens insidiously over several years. While all these symptoms have some day-to-day variation, episodes of marked deterioration in symptoms and functional capacity, known as acute exacerbations, occur periodically and more frequently as COPD progresses.

Overlap of asthma and COPD

The overarching term chronic obstructive pulmonary disease (COPD) may include patients with a variety of distinct clinical and other features who present differently and respond differently to treatment—these are referred to as 'clinical phenotypes'. Examples of phenotypes include patients with predominant breathlessness, those with recurrent exacerbations and those with asthma 'overlap.' Many patients with COPD report a history of asthma.

Clinical features favouring COPD include:

- onset after age 40
- symptoms persisting after several weeks or months of inhaled corticosteroid (ICS) treatment
- persistent airflow limitation
- heavier tobacco smoke exposure.

Clinical features favouring asthma include:

- onset before age 20
- significant day-to-day variability in symptoms and airflow limitation
- symptoms worse at night or in the early morning
- normal lung function between symptoms
- previous medical diagnosis of asthma
- family history of asthma or atopy
- seasonal variability in symptoms
- spontaneous improvement in symptoms.

There is evidence that patients with overlap of COPD and asthma experience more rapid disease progression than those with either disease alone. These patients have worse health-related quality of life, and experience more frequent and severe respiratory exacerbations. This is despite younger age and reduced lifetime smoking exposure when compared to those with COPD alone.

The evidence base for managing patients with overlap of COPD and asthma is limited because they are commonly excluded from clinical trials. ICS have a key role in preventing asthma-related deaths in patients with asthma, and long-acting bronchodilators have a significant role in COPD management; therefore, it would be expected that they could be used in patients with overlap of asthma and COPD. Avoid monotherapy with either ICS or long-acting bronchodilators in this group of patients.

The approach to using ICS in asthma is to establish the minimal effective dose; however, in COPD clinical trials have used only medium to high doses and the effect of lower doses has not been adequately explored.

Assessment

Overview

A medical history and clinical examination may suggest a diagnosis of chronic obstructive pulmonary disease (COPD), but cannot reliably predict the presence of airflow limitation. Spirometry is essential to determine whether COPD is the probable cause of respiratory symptoms. Consider the degree of breathlessness induced by daily activities and frequency of exacerbations when evaluating overall disease severity. Also assess for the presence of comorbidities and sequelae of COPD (eg cor pulmonale, hypoxaemia), which may require additional treatment.

Measuring lung function

The spirometric abnormality required to diagnose COPD is a post-bronchodilator forced expiratory volume in 1 second (FEV_1) to forced vital capacity (FVC) ratio less than 0.7 (ie an obstructive pattern). This definition is widely accepted because of its practicality, although its use may lead to overdiagnosis in the older person (as FEV_1 declines more rapidly with age than does FVC) and underdiagnosis in younger adults.

More extensive lung function testing, which is not availablein Fiji, will often also show gas trapping (increased residual volume) and a diffusion defect (decreased diffusing capacity for carbon monoxide [DLCO]).

Maintenance management of chronic obstructive pulmonary disease

General principles

The goals of managing stable chronic obstructive pulmonary disease (COPD) are to:

- reduce symptoms
- reduce frequency and severity of exacerbations and consequent long-term decline in lung function
- improve exercise tolerance
- improve health-related quality of life
- slow disease progression.

Guidelines for COPD management recommend a stepwise escalation of therapy based on disease severity. In stable COPD, first give attention to:

- smoking cessation
- encouragement of physical activity
- pulmonary rehabilitation

- current immunisation.
- nutrition

For patients with few symptoms, the above interventions may be adequate initially.

Introduction of further therapy is guided by ongoing assessment and response to initial treatment. As the disease progresses, introduce drug treatment to target day-to-day symptoms (primarily breathlessness) and to prevent exacerbations. For symptom control there should be a stepwise approach to drug therapy until adequate control has been achieved. This is summarised in the figure below and discussed in more detail with dosing regimens given below.

Some drug treatments are also effective in preventing deterioration, either by decreasing exacerbations and hospitalisations, attenuating decline in quality of life, or both. The effect of drug treatment on mortality remains unclear.

Smoking cessation

When managing COPD, prioritise smoking cessation to prevent or limit lung damage. Tobacco smoking is the main cause of COPD, and smoking cessation is the only intervention that has been shown to improve the natural history of COPD (apart from oxygen therapy in those with severe hypoxaemia.

Physical activity

Many patients with COPD have a sedentary lifestyle and it is important to encourage an increase in regular physical activity. Evidence suggests that performing some form of regular activity may lower the risk of hospitalisation for patients with COPD.

Pulmonary rehabilitation

Pulmonary rehabilitation is extremely effective therapy; it improves quality of life and exercise capacity in patients at all stages of COPD severity. There is increasing evidence that it reduces frequency of admissions and length of hospital stay.

Most pulmonary rehabilitation programs contain four elements:

- exercise training
- education
- behaviour modification
- outcome assessment.

The key components of exercise training are aerobic training and strength training of the upper and lower limbs. Flexibility exercises are often included. Breathing techniques and positioning are taught, which assist patients to recover from episodes of breathlessness. Targeted inspiratory muscle training may be used for some patients.

Benefits can be sustained even after a single pulmonary rehabilitation program. The longer a program continues, the more effective the results. Benefit wanes after a rehabilitation program ends, but if exercise training is maintained at home the patient's health status remains above pre-rehabilitation levels.

Immunisation

Patients with COPD should receive an influenza vaccine every year, and guidelines also recommend up-to-date pneumococcal vaccination for all people with COPD. Neither currently available on Fiji EML.

Nutrition

Although the evidence for benefit of nutritional supplements in underweight patients with COPD is limited, high-calorie supplements might be considered in malnourished patients.

Figure 4: Stepwise management of stable COPD

	MILD	MODERATE	SEVERE
General management	**Reduce risk factors**: avoid exposure to risk factors (eg tobacco smoke, air pollution), support smoking cessation, recommend pneumococcal and annual influenza vaccination		
	Optimise function: encourage regular exercise and physical activity, review nutrition, provide education about COPD, develop a GP management plan and written COPD action plan, and undertake regular review		
	Optimise treatment of comorbidities, especially cardiovascular disease, anxiety and depression, osteoporosis and lung cancer		
	Refer symptomatic patients for pulmonary rehabilitation		
		Initiate advance care planning	
Stepwise drug management	Start with as-needed short-acting bronchodilator therapy		
	Add regular long-acting bronchodilator monotherapy (with a LAMA *non-EML* or LABA *non-EML*)		
	Consider regular long-acting bronchodilator dual therapy (with a LAMA *non-EML* and LABA *non-EML*) if not controlled with monotherapy		
	Consider regular LAMA *non-EML*, LABA *non-EML* and ICS triple therapy if **both** the following apply: • the patient has had a severe exacerbation (requiring hospitalisation) or at least two moderate exacerbations in the past 12 months, and • the patient has significant symptoms despite dual therapy with a LAMA *non-EML* and LABA *non-EML*		
Assess and optimise inhaler device technique at each visit			
Additional therapies for severe COPD			Consider: • domiciliary oxygen therapy for hypoxaemia • long-term noninvasive ventilation for hypercapnia • palliative care services • surgery or bronchoscopic interventions

cont...

COPD = chronic obstructive pulmonary disease; GP = general practitioner / primary care physician; ICS = inhaled corticosteroid; LABA = long-acting beta$_2$ agonist; LAMA = long-acting muscarinic antagonist.

Note 1: Precautions:

- monotherapy with a LABA should not be used for patients with asthma or patients with overlap of asthma and COPD
- if starting a LAMA, discontinue ipratropium (a short-acting muscarinic antagonist [SAMA])
- if starting a fixed-dose LABA+LAMA combination inhaler, discontinue existing LABA and/or LAMA single-drug inhalers
- if starting a fixed-dose ICS+LABA combination inhaler, discontinue existing LABA single-drug inhaler.

Adapted with permission from: Yang IA, George J, McDonald CF, McDonald V, O'Brien M, Smith B, Zwar N, Dabscheck E. The COPD-X Plan: Australian and New Zealand Guidelines for the management of Chronic Obstructive Pulmonary Disease 2021. Version 2.63, February 2021.

LAMA (long-acting antimuscarinic antagonists) and LABAs (long-acting beta$_2$ agonists) were not available on the Fiji EML at the time of publishing. Some options may be available from private pharmacies. Alternatively, consider other management options discussed under 'Drug treatment' in the next section.

Current international recommendations are shown in Figure 4, but not all treatments are available on the Fiji EML. At the time of publishing, treatment options on the Fiji EML were limited to SABA and ICS. ICS/LABA and SAMA were available in the private sector; LABAs and LAMAs as single ingredient preparations may be difficult to obtain and are costly.

Drug treatment

Overview

The aims of drug treatment in chronic obstructive pulmonary disease (COPD) are to relieve symptoms and to prevent deterioration, either by reducing frequency of exacerbations or by reducing decline in quality of life, or both. Bronchodilators available overseas include short-acting beta$_2$ agonists (SABAs) and long-acting beta$_2$ agonists (LABAs) as well as short-acting muscarinic antagonists and long-acting muscarinic antagonists (LAMAs). Even in the absence of a demonstrable improvement in forced expiratory volume in 1 second (FEV_1) they can reduce the gas trapping that is a feature of COPD, which may improve breathlessness and exercise capacity. As well as improving symptom control, both LAMAs and LABAs have been shown to reduce exacerbations and hospitalisations.

Despite the relative insensitivity of the inflammatory response in COPD to corticosteroids, inhaled corticosteroids (ICS) are recommended for patients with moderate to severe COPD, especially in those with recurrent exacerbations. ICS give benefits of reduced exacerbations and improved quality of life. However, these small additional benefits must be balanced against an increased risk of pneumonia and local adverse effects (dysphonia, upper airway candidiasis).

Regularly review the patient's medication and either continue or stop drugs based on treatment response and tolerability. Check inhaler technique and adherence at each visit; up to 90% of patients use their devices incorrectly. It is important that both the patient and treating doctor are clear about goals of treatment, ie symptom control or longer-term outcomes (such as prevention of exacerbations and/or hospitalisations), or both.

Inhaled bronchodilators

For the long-term treatment of COPD, bronchodilators are recommended for symptom relief, to increase physical capacity and to improve quality of life. Long-acting bronchodilators have also been shown to reduce exacerbations.

The preferred route of administration is by inhalation, which requires correct delivery device technique. Evidence suggests that a MDI with spacer is as effective as a nebuliser; however, the appropriate method of inhalation depends on the patient's therapeutic needs, characteristics and preference.

Many symptomatic patients get significant relief from bronchodilator therapy. Benefit can only be assessed by a therapeutic trial with symptom control or quality of life as endpoints, which may take several weeks.

Short-term response to an inhaled bronchodilator in the setting of an acute exacerbation does not necessarily predict response to regular long-term use. Conversely, some patients who do not have a short-term response may still benefit from a long-term trial. This is because relief of gas trapping (not measured routinely), which correlates well with relief of dyspnoea, may occur in the absence of a significant change in forced expiratory volume in 1 second (FEV_1). It is difficult to determine the length of time a long-acting bronchodilator should be trialled to assess its impact on frequency of exacerbations; depending on the patient's usual frequency of exacerbations, the trial may need to continue for months or up to a year.

Short-acting bronchodilator therapy

Short-acting bronchodilator therapy is used as required to provide short-term symptom relief for patients with COPD. There is no evidence to suggest that short-acting bronchodilator drugs reduce the rate of decline in lung function or have any effect on survival. For patients with mild and infrequent symptoms, a combination of general measure and short-acting $beta_2$ agoinist (SABA) bronchodilator therapy may be adequate.

Patients with poor inhaler technique should use a pressurised metered dose inhaler (MDI) with a spacer, which improves lung deposition of the drug. Use:

> salbutamol 100 micrograms per puff (actuation), 2 puffs by inhalation via MDI (with or without spacer), as needed.

Ipratropium is not usually used for **symptom relief** in COPD—it is contraindicated in

patients taking a long-acting muscarinic antagonist (LAMA), is more expensive than a SABA, and may increase the risk of cardiovascular events.

Generally, a nebuliser is only necessary for patients who are unable to use a MDI (with or without a spacer).

Assess response 3 months after starting treatment. In patients who remain symptomatic, check inhaler technique before considering stepping up therapy.

Long-acting bronchodilator therapy (LAMA or LABA)

Overview

Long-acting bronchodilators provide symptomatic relief of breathlessness and improve exercise capacity; they may also reduce frequency and severity of exacerbations and improve quality of life. However, they are expensive, and are currently not included on the Fiji EML.

Short-acting bronchodilators as 'rescue' medication are usually used in conjunction with long-acting bronchodilators. Long-acting inhaled bronchodilators include long-acting beta$_2$ agonists (LABAs) and long-acting muscarinic antagonists (LAMAs). These can be used either separately as monotherapy, or in combination for patients experiencing persistent breathlessness. Monotherapy with a LABA should not be used for patients with overlap of asthma and COPD.

LAMAs are not available on the Fiji EML but there are a number of products available globally, including tiotropium 10 or 18 micrograms by inhalation via DPI, daily OR tiotropium 5 micrograms by inhalation via mist inhaler, daily.

Ipratropium (a short-acting muscarinic antagonist) is available to be purchased privately and could be used **regularly** (6-hourly):

> ipratropium 21 micrograms, 2 puffs by inhalation via MID (with or without spacer), 6-hourly [non-EML]

LABAs currently not available on EML, only available in combination with ICS in private sector (see next section, ICS).

If patients experience frequent exacerbations, consider adding an inhaled corticosteroid (ICS) to their treatment regimen.

Inhaled corticosteroids

The aims of treatment with inhaled corticosteroids (ICS) in COPD are to reduce exacerbation rates and slow decline in health status, not to improve lung function per se. The effect of ICS on mortality is uncertain. Response to oral corticosteroids does not predict response to ICS; do not use oral corticosteroids to identify which patients may benefit from ICS.

Clinical trials have observed an increased risk of pneumonia associated with ICS use in patients with COPD.

Smoking adversely affects response to ICS; advice patients who continue to smoke that the benefits of ICS treatment is likely to be reduced.

ICS are indicated as add-on therapy for patients with both:

- a forced expiratory volume in 1 second (FEV_1) less than or equal to 50% predicted
- two or more exacerbations requiring treatment with antibiotics or oral corticosteroids in a 12-month period.

ICS are also prescribed as add-on treatment for patients with documented evidence of responsiveness to ICS on the grounds of spirometry or functional status. ICS may be indicated for patients with overlap of asthma and COPD.

Doses of ICS (inhaled corticosteroid) recommended in COPD are the same as for the prevention of asthma.

Table 21: Corticosteroid-based-inhalers available in Fiji for COPD

Drug	**Dosage** [Note 1]		
	Low	**Medium**	**High** [Note 2][Note 3]
beclometasone	50 or 100 micrograms twice daily	200 micrograms twice daily	300 or 400 micrograms twice daily
budesonide (including combinations with LABA) Non-EML	100 or 200 micrograms twice daily	400 micrograms twice daily	600 or 800 micrograms twice daily maximum 2400 micrograms daily in divided doses
fluticasone propionate (including combinations with LABA) Non-EML	50 or 100 micrograms twice daily	125 to 250 micrograms twice daily	500 micrograms twice daily maximum 1000 micrograms twice daily

COPD = chronic obstructive pulmonary disease; DPI = dry powder inhaler; LAMA = long-acting muscarinic antagonist; SABA = short-acting $beta_2$ agonist; MDI = pressurised metered dose inhaler

Note 1: For information about delivery devices, including links to videos and patient handouts, see 'Inhalational drug delivery devices,' page 197.

Note 2: Unless a maximum daily dose is specified, the high-dose regimen is also the maximum daily dose.

Note 3: Except for short periods, refer patients considered for high doses to a specialist.

To minimise the risk of oropharyngeal candidiasis and systemic corticosteroid absorption, advise patients to rinse their mouth with water and spit out straight after using ICS. Patients using a pressurised metered dose inhaler (MDI) should also be advised to use a spacer, which decreases oropharyngeal deposition of the inhaled corticosteroid, thereby reducing systemic absorption, oropharyngeal candidiasis and dysphonia.

Oral corticosteroids should preferably not be used for maintenance therapy in COPD due to increased risk of adverse effects, including weight gain and the development of diabetes, cataracts and osteoporosis.

Oxygen

Long-term continuous oxygen therapy, usually delivered by a concentrator, offers survival benefits for patients with COPD and partial pressure of oxygen (PaO2) of 55 mmHg or less, or those with PaO2 55 to 59 mmHg with cor pulmonale, pulmonary hypertension or polycythaemia. It needs to be used for at least 18 hours a day to maximise benefits.

Oxygen concentrators are expensive and are generally not available for use in Fiji.

Theophylline

Theophylline may be helpful in some patients with COPD. However, at standard doses it has potential for significant drug interactions and adverse effects. It should be considered only for patients in whom other treatment has failed to control symptoms adequately, or for patients who are unable to use inhaled therapy.

Studies have suggested low-dose theophylline (eg 150 mg to 300 mg SR daily) may have anti-inflammatory effects and may reduce exacerbation frequency. However, the role of theophylline for patients on maximal bronchodilator and corticosteroid therapy is unclear.

Theophylline has a narrow therapeutic range, ideally blood levels should be monitored but this is currently unavailable in Fiji. Educate patients about common symptoms of toxicity such as nausea, vomiting, diarrhoea, tremor or palpitations, and to seek medical attention if they experience any of these.

Theophylline should only be initiated by a specialist and patients must have regular reviews.

Oral mucolytics

Over-the-counter oral mucolytics (usually bromhexine) are widely used by patients with COPD. They have not been studied for efficacy in COPD but appear to be safe.

Other management considerations

Comorbidities and complications

A number of conditions that commonly coexist with chronic obstructive pulmonary disease (COPD) have implications for diagnosis and/or management.

Cardiovascular disease

Patients with COPD are at high risk of cardiovascular disease. Diagnosing cardiovascular disease in COPD is made more difficult by similar presenting features, which in both cases may include breathlessness, fatigue and even chest discomfort.

Managing cardiovascular disease coexisting with COPD can also be difficult; beta blockers have proven mortality benefits in cardiovascular disease but their use remains low in patients with COPD because of the concern about acute bronchospasm, particularly in those with overlap of asthma and COPD. Reviews suggest that cardioselective beta blockers are generally safe and well tolerated for patients with COPD, but a cautious approach with low dose initiation and gradual up-titration is recommended.

Anxiety and depression

Anxiety, including panic attacks, and depression occur frequently in patients with COPD and adversely affect prognosis, particularly in those who are admitted to hospital. Comprehensive pulmonary rehabilitation can significantly reduce symptoms of both anxiety and depression; there are also promising results with both cognitive behavioural therapy and a collaborative care model. More severe symptoms may merit pharmacotherapy.

Osteoporosis

Patients with COPD have a higher than normal frequency of bone fracture, and their bone mineral density is on average 10% lower than control patients. Identification and management of those at risk should be undertaken using current guidelines for treatment of osteoporosis.

Diabetes

Patients with COPD are at increased risk of developing type 2 diabetes. Patients with COPD and comorbid diabetes are also at increased risk of diabetes complications if using high-dose inhaled corticosteroids (ICS). Review maintenance treatment for these patients regularly to ensure that minimally effective ICS doses are used, and blood glucose control is closely monitored.

Treatment with short courses of oral corticosteroids in these patients should be limited. It is important to optimise the balance between benefits of treatment and potential short- and long-term adverse effects. Anti diabetic therapy may need to be escalated during the corticosteroid course and subsequently de-escalated.

Pulmonary hypertension and cor pulmonale

Mild-to-moderate pulmonary hypertension is a common complication of COPD that is associated with increased risk of exacerbation, worsened quality of life and increased mortality. Pulmonary vascular remodelling is thought to result from the combined effects of hypoxia, inflammation and loss of capillaries in severe emphysema.

Despite known benefits in idiopathic pulmonary hypertension, there is no evidence to date that pulmonary vasodilator drugs (eg endothelin receptor antagonists, phosphodiesterase type-5 [PDE5] inhibitors) are beneficial in pulmonary hypertension complicating COPD. These drugs potentially may even cause harm by worsening ventilation perfusion mismatch and, consequently, hypoxaemia.

Cor pulmonale is defined as right ventricular hypertrophy and dilation secondary to lung disease; it should be considered if a patient with COPD has:

- peripheral oedema
- raised jugular venous pressure
- a parasternal heave
- a loud pulmonary second heart sound.

Oedema associated with cor pulmonale can usually be controlled with diuretic therapy. Manage right heart failure associated with cor pulmonale according to usual recommendations for the treatment of heart failure.

Prognosis, palliative care and advance care planning

The course of COPD is often punctuated by recurrent exacerbations, which may require hospitalisation and consideration of assisted ventilation. Hospitalisation for acute exacerbation increases subsequent mortality risk. As the disease progresses a palliative approach to care may be appropriate. Determining prognosis in end-stage COPD is difficult; however, characteristics that should trigger discussions about a palliative approach to care, advance care planning, and end-of-life issues include:

- forced expiratory volume in 1 second (FEV_1) less than 25% of predicted
- oxygen dependence
- respiratory failure
- heart failure or other comorbidities
- weight loss or cachexia
- decreased functional status
- increasing dependence on others
- advanced age.

Ideally, end-of-life discussions, including resuscitation and intubation wishes and advance care planning, should occur in an outpatient setting when the patient's condition is relatively stable.

Patients with severe COPD who are in the palliative care phase may benefit from low-dose opioids, which reduce the sensation of breathlessness without significantly depressing respiration.

Specialist referral

Patients with chronic obstructive pulmonary disease (COPD) who have persistent dyspnoea despite optimised therapy may benefit from specialist referral. Specialist knowledge and investigations can confirm the diagnosis of COPD and identify patients with either asthma oroverlap of asthma and COPD. In addition, specialist input assists in differentiating COPD from other airway diseases or occupational exposures that may cause airway narrowing and/or airway hyperresponsiveness.

Ongoing monitoring and review

Patients with chronic obstructive pulmonary disease (COPD) should be reviewed regularly; frequency of review depends on various factors including patient symptoms and history of exacerbations. A summary of suggested regular review for patients with COPD in primary care is outlined below.

Table 22: Regular review of patients with COPD based on severity

	Severity of COPD	
	Mild and moderate [Note 1]	**Severe** [Note 1]
Minimum frequency of review [Note 2]	at least annually	at least twice per year
Clinical assessment	• smoking status and desire to quit • adequacy of symptom control: • breathlessness • exercise tolerance • exacerbation frequency • presence of complications • effects of each drug treatment • inhaler technique • adverse effects of treatment • adherence • use of written COPD action plan if appropriate • presence of psychiatric comorbidities (eg depression, anxiety) • need for referral to specialist physician and therapy services	clinical assessment as for mild and moderate COPD, plus: • presence of cor pulmonale • possible coexisting cardiovascular disease (including heart failure, atrial fibrillation, ischaemic heart disease), particularly if there is evidence of recent COPD deterioration • patient's nutritional state • need for social services and occupational therapy input • need for end-of-life discussion and advance directives
Measurements	• FEV_1 and FVC if available (Note 3) • BMI	measurements as for mild and moderate COPD, plus: • arterial blood gases if SpO2 is less than 92%

BMI = body mass index;; COPD = chronic obstructive pulmonary disease; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; SpO_2 = oxygen saturation measured by pulse oximetry

Note 1: See text for typical symptoms and FEV_1.

Note 2: More frequent opportunistic assessment of factors such as inhaler technique, smoking status, symptom control and adverse effects of treatment is encouraged.

Note 3: Perform spirometry annually (if available) in mild, moderate and severe COPD.

Management of acute exacerbations of chronic obstructive pulmonary disease

An acute exacerbation of chronic obstructive pulmonary disease (COPD) is characterised by the following features, beyond day-to-day variations:

- increasing dyspnoea
- reduced exercise tolerance
- tachypnoea.

An exacerbation may also be associated with:

- increasing cough frequency
- increasing sputum volume and/or purulence, with or without fever
- right heart failure, manifesting as ankle oedema.

The overlap between cardiac and pulmonary reasons for acute deterioration is common; diagnosis is made more difficult by similar presenting features, which in both cases may include breathlessness, fatigue and even chest discomfort.

Exacerbations are associated with considerable morbidity, mortality and healthcare costs. Exacerbations become more frequent as the severity of COPD worsens. Following hospitalisation for an exacerbation, quality of life and lung function decline and patients are at risk for further serious exacerbations. Reducing exacerbations is therefore a primary goal of treating COPD.

Triggers include viral and bacterial infections as well as environmental pollutants, heart failure and pulmonary embolism. Prompt treatment with short-acting bronchodilators, antibiotics as appropriate and corticosteroids hastens resolution and reduces need for hospitalisation. Noninvasive ventilatory support is indicated for hypercapnic respiratory failure and is effective in avoiding intubation and reducing risk of death. An admission to hospital with an exacerbation of COPD is a sentinel event that should trigger review of current management, including preventive therapies and consideration of advance care planning. Clarify patient wishes concerning use of noninvasive and invasive ventilation should the need arise.

Assessing severity

Objective assessment of the severity of a chronic obstructive pulmonary disease (COPD) exacerbation can be difficult; it is based largely on the underlying severity of the patient's condition, in addition to other aspects of their medical history, clinical signs and investigations – see Table below. The combination of these factors determine whether the exacerbation can be managed in an outpatient setting, or if the patient should be referred to hospital for assessment with or without admission. Additional indicators are listed in the Box below.

Table 23: Considerations in assessing severity of a COPD exacerbation

Assessment	Details
Medical history	• severity of underlying COPD • duration of worsening or new symptoms • number of previous exacerbations (total; note how many required hospitalisation) • comorbidities • current treatment regimen • previous need for mechanical ventilation • recent increased oxygen requirements (if applicable) • recent degree of difficulty with activities of daily living
Signs of severity	• use of accessory respiratory muscles • paradoxical chest wall movements • worsening or new-onset central cyanosis • development of peripheral oedema • haemodynamic instability • deteriorated mental status
Additional investigations, if appropriate:	
pulse oximetry [Note 1]	useful for tracking and/or adjusting supplemental oxygen therapy
chest X-ray	useful to exclude alternative diagnoses
electrocardiogram	may aid in diagnosis of coexisting cardiac problems
full blood count	may identify polycythaemia, anaemia, leucocytosis
presence of purulent sputum	presence can be sufficient indication for starting antibiotic therapy
biochemical test abnormalities	electrolyte disturbances and hyperglycaemia can be associated with exacerbations; however, they can also be due to associated comorbidities
spirometry	not recommended to assess an exacerbation [Note 2]

COPD = chronic obstructive pulmonary disease

Note 1: Measuring arterial blood gases is vital if coexistence of acute or acute-on-chronic respiratory failure is suspected. Assessing the acid–base status is necessary before initiating ventilatory support.

Note 2: Spirometry should ideally be performed during the hospital admission to confirm diagnosis and provide an estimate of disease severity.

Source: Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Bethesda, MD: GOLD; updated 2014. <www.goldcopd.org>

Box 5: Possible indicators for hospital assessment or admission in COPD exacerbations

The following may be indicators for hospital assessment or admission of a patient with an exacerbation of COPD [Note 1]:

- Marked increase in intensity of symptoms (such as sudden development of resting dyspnoea).
- Acute exacerbation characterised by increased dyspnoea, cough or sputum production, plus one or more of the following:
  - inadequate response to ambulatory management
  - inability to walk between rooms when previously mobile
  - inability to eat or sleep because of dyspnoea
  - cannot manage at home / insufficient home support
  - high risk comorbidity condition – pulmonary (eg pneumonia) or non-pulmonary
  - altered mental status suggestive of hypercapnia
  - worsening hypoxaemia or cor pulmonale
  - newly occuring arrhythmia

COPD = chronic obstructive pulmonary disease

Note 1: Local resources need to be considered.

Adapted with permission from: Yang IA, George J, McDonald CF, McDonald V, O'Brien M, Smith B, Zwar N, Dabscheck E. The COPD-X Plan: Australian and New Zealand Guidelines for the management of Chronic Obstructive Pulmonary Disease 2021. Version 2.63, February 2021

Treatment

Inhaled bronchodilators

Treat patients with an acute exacerbation of chronic obstructive pulmonary disease (COPD) with a bronchodilator. Most studies suggest that $beta_2$ agonists (salbutamol) and the antimuscarinic drug ipratropium work equally well, although the onset of effect of salbutamol is more rapid than that of ipratropium. Some patients may respond better to one type of medication than the other; tailor treatment to individual response.

In all patients using a pressurised metered dose inhaler (MDI), particularly those with poor inhaler technique, using a spacer improves lung deposition of the aerosol. A MDI with spacer is likely to be as effective as nebulisation for patients with forced expiratory volume in 1 second (FEV_1) greater than 30% predicted, although evidence for this comes from studies in patients with asthma.

For initial treatment of an acute exacerbation of COPD, use:

salbutamol 100 micrograms per puff (actuation), up to 10 separate puffs by inhalation via MDI with spacer, repeated as needed

If a nebuliser is used in COPD, it should be driven by compressed air, to avoid excessive and potentially deleterious hyperoxygenation. If appropriate, use:

1. salbutamol 2.5 to 5 mg by inhalation via nebuliser, as needed

OR (except in patients taking a LAMA)

2. ipratropium 250 to 500 micrograms by inhalation via nebuliser, as needed.

If these do not control symptoms adequately, or if symptoms are severe, combining salbutamol and ipratropium at the doses given above may provide added benefits without compounding adverse effects.

If these treatment regimens do not control symptoms, or if medication is required more frequently than 3-hourly, the patient should seek medical attention and/or enact their written COPD action plan if they have one.

Systemic corticosteroids

A 5-day course of systemic corticosteroids is recommended for exacerbations that don't respond sufficiently to inhaled bronchodilators. Systemic corticosteroids shorten the duration of hospital admission and hasten return to previous lung function and symptom control. Patients with a written COPD action plan may have already started oral corticosteroids when they present for medical attention. Use:

prednisolone 30 to 50 mg orally, once daily in the morning for 5 days.

If oral medication cannot be tolerated, use:

hydrocortisone 100 mg IV, 6-hourly until oral intake tolerated.

Switch to oral therapy as soon as is practical, and continue for a total of 5 days (oral + IV).

Short courses of prednisolone (less than 2 weeks) can be stopped abruptly, or reduced to the patient's baseline dose, without the need for routine tapering.

Antibiotics

Antibiotics should **not** be used unless the patient has clinical signs of infection. The aim of treatment is to hasten recovery and not to eliminate colonising organisms. Do not use antibiotics other than those listed below, as they do not have superior efficacy. If antibiotics are indicated, empirically use:

amoxicillin 500 mg orally 8 hourly for 5 days

OR

doxycycline 200 mg orally for the first dose, then 100mg orally daily for a total duration of 5 days

OR

chloramphenicol 500 mg orally 6-hourly for 5 days

Use doxycyline or chloramphenicol (not amoxicillin) in patients with immediate or delayed penicillin hypersensitivity.

Oxygen therapy

If the patient is hypoxaemic, oxygen should be administered to maintain the arterial oxyhaemoglobin saturation at 88 to 92%. Patients with COPD are at risk of hypercapnic respiratory failure during an acute exacerbation; the principles of oxygen therapy in such patients are discussed in detail in the topic Oxygen Therapy. Keep supplemental oxygen to the minimum consistent with adequate oxygen saturation (SaO2), adequate peripheral oxygen delivery and stable partial pressure of carbon dioxide levels (PaCO2) and pH.

Ventilatory support

If available, noninvasive ventilation is the standard of care for patients with acute exacerbations of COPD associated with hypercapnic respiratory failure and acidosis despite the following:

- optimal drug therapy
- treatment of other complications (eg pneumothorax)
- attempts to minimise inspired oxygen while maintaining adequate oxygen saturation (SpO2) at 88 to 92% or more.

Noninvasive ventilatory support often avoids the need for intubation and has been shown to reduce mortality and length of hospital stay. After an episode of acute hypercapnic respiratory failure treated with noninvasive ventilation, patients are at high risk of readmission and life-threatening events during the following year.

Management in an intensive care unit should be considered and is mandated if invasive ventilation is required.

Cough

Introduction

Cough is one of the most common reasons for presentation to healthcare providers. In children, cough is a major source of parental anxiety. Differential diagnosis to identify the underlying pathology is important. However, once the most common causes have been eliminated, identifying an underlying condition can be difficult and complex.

Cough is generally classified according to its duration before presentation, because this assists in the differential diagnosis. In children, an acute cough is one that has been present for up to 2 weeks; if it has lasted for 2 to 4 weeks it is considered prolonged acute cough, and anything longer is considered chronic cough. In adults, acute cough is one that has been present for up to 2 weeks; if it has lasted for more than 8 weeks it is considered chronic persistent cough. Any repetitive cough can cause laryngeal irritation and inflammation, leading to persistence of the urge to cough.

A summary of common and/or important causes of cough are outlined in the table below

Alarm symptoms and findings in cough are listed in the box below. Cough can also affect the patient's quality of life through the impact of adverse consequences of cough, such as urinary incontinence in women, headaches, vomiting, rib fracture and sleep disturbance. Consider referral to a specialist if a patient's cough is proving resistant to simple therapies, appears excessively prolonged or if the patient has any of the alarm symptoms and findings listed below.

Table 24: Summary of common and/or important causes of cough

Cause [Note 1]	Comments
Acute cough [Note 2]	
acute bronchitis	Commonly follows viral upper respiratory tract infection; in patients without underlying airway disease, sputum is not a good guide to the need for antibiotics.
asthma and asthma-like syndromes	Uncontrolled asthma is a common cause of cough, but it is unusual for cough to be the sole symptom
drug-induced cough	Common precipitants include ACEIs and beta blockers; there is not always a close temporal relationship between starting a drug and development of cough.
inhaled foreign body	Sudden onset of cough, particularly if cough started while eating or, in a child, while playing with small objects.
Chronic cough [Note 3] [Note 4]	

cont...

postinfective cough	Follows an acute viral respiratory tract infection; may last up to 8 weeks.
persistent bacterial bronchitis	The most common diagnosis in a child with purulent sputum and cough lasting for more than 4 weeks.
Pertussis	Consider if cough is persistent and paroxysmal, and lasting for 2 to 6 weeks or more
gastro-oesophageal reflux or laryngopharyngeal reflux	Consider if patient has symptoms such as heartburn or water brash, or if cough is worse at night or after eating specific foods; diagnosis often made clinically or following a response to empirical treatment.
upper airway cough syndrome (previously called postnasal drip)	May be associated with excessive mucus production (eg due to allergic or nonallergic rhinopathy), or with increased perception of normal volumes of postnasal mucus.
upper airway dysfunction (vocal cord dysfunction)	May be triggered by any other cause of cough and worsened by irritants such as gastro-oesophageal reflux, exposure to smoke or fumes, or excessive use of the voice.
obstructive sleep apnoea	Inflammation of the pharynx from snoring may contribute to cough.
psychogenic or habit cough	Particularly in children; cough not present during the night.
inhaled foreign body	Sudden onset of cough, particularly if cough started while eating or, in a child, while playing with small objects.
tuberculosis	Usually presents with persistant cough with or without sputum, fever, night sweats and/or weight loss, shortness of breath, not responding to antibiotics and may have contact history with tuberculosis. Refer to the Fiji Tuberculosis Manual.
lung or laryngeal cancer	Consider in smokers older than 45 years with a new cough, altered cough, or cough with voice disturbance.
recurrent aspiration	Aspiration-prone people include patients with Parkinson's disease, stroke, dementia, COPD, impaired consciousness, or neuromuscular disorders affecting bulbar muscles.
chronic bronchitis	Productive cough, occurring every day for at least 3 months, at least 2 years in a row; occurs in the majority of heavy smokers (more than 20 pack years [Note 5]).
bronchiectasis	Productive cough and frequent chest infections

cont...

interstitial lung disease	Dry cough, often associated with shortness of breath
cystic fibrosis	Productive cough associated with gastrointestinal symptoms or failure to thrive
drug-induced cough	Common precipitants include ACEIs and beta blockers; there is not always a close temporal relationship between starting a drug and development of cough.

ACEIs = angiotensin converting enzyme inhibitors; COPD = chronic obstructive pulmonary disease

Note 1: For detailed advice on diagnosis and assessment of cough in children and adults, see the CICADA guidelines <www.mja.com.au/journal/2010/192/5/cicada-cough-children-and-adults-diagnosis-and-assessment-australian-cough>.

Note 2: Acute cough is present for up to 2 weeks in children and adults.

Note 3: Chronic cough is present for more than 4 weeks in children and more than 8 weeks in adults.

Note 4: Some of these diagnoses should also be considered as potential causes of acute cough, as the patient may present soon after onset of cough.

Note 5: Pack years is calculated using the formula (years of smoking × cigarettes per day) / 20; see also: http://smokingpackyears.com.

Box 6: Alarm symptoms and findings in adults with cough

Consider additional investigations or referral to a relevant specialist if an adult with a cough has any of the following symptoms or findings:

- hamoptysis
- smoker who
  - has a greater than 20 pack-year smoking history [Note 1] and a new or altered cough
  - is older than 45 years and has a new or altered cough, or cough with voice disturbance
- prominent dyspnoea, especially at rest or at night
- recurrent or chronic sputum production
- hoarseness
- systemic symptoms (eg fever, weight loss, vomiting, oedea)
- GORD associated with weigh loss, anaemia, haematemesis or malaena, vomiting, dysphagia (difficulty swallowing), odynophagia (painful swallowing)
- GORD not responding to empirical treatment
- Recurrent pneumonia
- Abnormal clinical respiratory examination
- Abnormal chest X-ray

GORD = gastro-oesophageal reflux disease.

Note 1: Pack years is calculated using the formula (years of smoking x cigarettes per day) / 20.

General principles

The initial step in treating cough is identification and specific management of the cause(s) of cough. However, persistent cough may become self-perpetuating because the mucosa of the upper airways becomes denuded and inflamed due to the coughing. The damaged mucosa is more sensitive and any exposure to external triggers may lead to renewed bouts of coughing. For many patients with chronic cough, nondrug interventions are useful to break this self-perpetuating cycle. For patients with persistent productive cough, strategies to facilitate mucus clearance may be useful. Few patients with either acute or chronic cough require antibiotic treatment.

For patients with cough of any cause, nonspecific interventions can help to reduce symptoms and decrease laryngeal irritation while any specific causes are being identified and (where relevant) treated. For patients with a dry cough, these nonspecific interventions focus on reducing the urge to cough, whereas for patients with a wet or productive cough, the focus is on facilitating sputum clearance.

For cough associated with respiratory tract infection, explain to patients that antibiotics:

- are only rarely needed, because most respiratory tract infections are viral
- can cause adverse effects such as diarrhoea, thrush and allergic reactions
- can cause antibiotic resistance if they are used when not needed, making future infections harder to treat.

Providing a 'prescription' with advice about nonspecific strategies for dealing with acute respiratory tract infection may be useful to reduce patient expectation for antibiotics.

After a trial of any drug treatment for cough, assess the response and stop the treatment if it is not effective. In severe or intractable cough, consider referral.

Nondrug interventions

Environmental factors

When relevant, advise patients to avoid exposure to environmental factors that may be contributing to or worsening their cough. This includes exposure to environmental tobacco smoke and other inhaled irritants, and to cold dry air.

Vocal hygiene

Vocal hygiene measures for patients with a dry cough are aimed at reducing further irritation to the larynx, which may be perpetuating the urge to cough. These strategies include:

- avoiding overuse of the voice
- avoiding smoky or polluted environments
- avoiding clearing the throat and minimising coughing; for example by taking sips of water with a hard swallow
- having a family member draw attention to unwitting habitual coughing or throat-clearing (not advised for children because it tends to reinforce the habit)

Sputum clearance

For patients with a chronic productive cough, training in 'Active Cycle of Breathing' techniques can improve the effectiveness of sputum clearance by moving secretions towards the mouth so they can be cleared. This can reduce laryngeal trauma from repetitive coughing.

Drug treatment

For cough associated with a self-limiting respiratory tract infection (eg acute bronchitis, acute rhinosinusitis), many patients have an expectation of treatment with antibiotics. Effective communication with the patient or carer about the role of antibiotics is essential. The discussion should address misconceptions about the effectiveness of antibiotic therapy and the expectation of an antibiotic prescription.

Caution for children

Cough and cold medicines for children have minimal, if any, evidence of efficacy, are costly, and can cause harm. Cough and cold medicines, including cough suppressants, antihistamines, decongestants and combination products, **should not be given to children younger than 6 years**. Caution should also be used when treating children aged 6 to 12 years.

Cough suppressants

Treatment with opioid cough suppressants (codeine, dextromethorphan, dihydrocodeine, pholcodine) may be considered in adults for a limited duration. However, cough suppressants have only marginal benefit over placebo, and the potential for addiction with opioids should be considered. Cough suppressants are not recommended for use in children.

If appropriate, use a cough suppressant for a limited duration in adults for short-term relief and to break the cough cycle.

Cough expectorants

The aim of mucolytic treatment is to reduce mucus viscosity and aid its expectoration. Oral bromhexine is marketed as a mucolytic, but evidence for efficacy is limited.

Antihistamines

Antihistamines have not been found to be more effective than placebo in relieving acute cough, although some clinicians recommend using a sedating antihistamine (eg promethazine) at night if dry cough disturbs sleep.

Proton pump inhibitors

If gastro-oesophageal reflux disorder (GORD) is considered likely (eg symptoms of heartburn or water brash, night-time choking without symptoms of obstructive sleep apnoea), an empirical trial of high-dose proton pump inhibitors may be used. Review response after 8 to 12 weeks and stop if there is no response. Also recommend lifestyle changes, such as avoiding caffeine and meals late at night.

Corticosteroids

For patients with symptoms of allergic or nonallergic rhinopathy, an empirical trial of intranasal saline (sodium chloride solution) and intranasal corticosteroids may be given for at least 1 month.

For patients with otherwise unexplained chronic cough, empirically trial low-dose inhaled corticosteroids and review response after 2 to 4 weeks. If the cough responds, this does not necessarily indicate a diagnosis of asthma. Measurement of lung function before and after the trial of inhaled corticosteroids will help to confirm a diagnosis of asthma. Cough due to eosinophilic bronchitis may also respond to inhaled corticosteroids.

Complementary medicines

Evidence for the benefit of honey in cough mainly comes from studies of children. There is some evidence of benefit in adults with cough, although more studies are needed. Honey may be trialled in adults with cough because it is safe and easily accessible.

There is minimal evidence for the effectiveness of the many complementary therapies sold over-the-counter for treatment of cough.

The expectorant senega with ammonia has no evidence of benefit in cough. Some complementary therapies such as echinacea have been associated with severe allergic reactions.

Acute bronchiolitis

Acute viral bronchiolitis is the most common lower respiratory tract infection in infants. It affects both the upper and lower airways and has a peak prevalence and severity in infants younger than 6 months. The diagnosis of bronchiolitis is clinical and based on a typical history of nasal discharge and fever, and examination findings of tachypnoea, increased work of breathing, widespread inspiratory crackles and expiratory wheeze. The diagnosis is usually limited to the first 12 months of life.

Babies born prematurely and children with congenital heart disease or undergoing treatment for haematological malignancy are at particular risk for severe disease. There is an increased risk in infants of parents who smoke and in male infants. Around 1% of infants are hospitalised for acute viral bronchiolitis. Respiratory syncytial virus is the most common associated virus, although other respiratory viruses such as rhinovirus, human metapneumovirus, parainfluenza and influenza may be involved.

Clinical presentation and severity

Do not routinely undertake chest X-rays for the diagnosis of bronchiolitis in children.

Bronchiolitis typically begins with an acute upper respiratory tract infection followed by onset of respiratory distress and fever and one or more of

- cough
- tachypnoea
- retractions
- widespread crackles or wheeze

Refer to Table 25 for assessment of severity)

Risk factors for more serious illness

- prematurity (gestational age < 37weeks)
- age less than 12 weeks
- low birth weight
- chronic lung disease
- congenital heart disease
- chronic neurological/neuromuscular disease
- immunodeficiency
- parent unable to care for child at home
- passive smoking
- daycare attendance

Infants with any of the above risk factors are more likely to deteriorate rapidly and require escalation of care. Consider hospital admission even if presenting early in illness with mild symptoms.

Table 25: Assessment of severity of bronchiolitis

This table is to provide guidance to stratify severity. The more symptoms the infant has in the mod-severe categories, the more likely the patient will develop severe disease.

	Mild	Moderate	Severe
Behaviour	Normal	Some/intermittent irritability	Increasing irritability and/or lethargy/ fatigue
Respiratory rate	Normal/mild tachypnoea	Increased respiratory rate	Marked increase or decrease in respiratory rate
Use of accessory muscles (respiratory distress)	nil to mild chest wall retraction	moderate chest wall and suprasternal retractions Moderate nasal flaring	Marked chest wall and suprasternal retractions Marked nasal flaring
SpO2/ oxygen requirement	> 92% (in room air)	90 – 92% (in room air)	< 90% (in room air) Hypoxemia, may not be corrected by O2 (Patients with congenital cardiac lesions may have low baseline saturations < 90%)
Apnoeic episodes	None	Brief, self-limiting apnoea	Frequent or prolonged apnoea
Feeding	Normal	SOB when feeding	Reluctant or unable to feed

SpO2 = oxygen saturation measured by pulse oximetry.

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Exam findings may change quickly due to varying clearance of obstruction. Reassess frequently to guide management.

Investigations

In most children with bronchiolitis, **no** investigations are required as they play no role in management. Investigations should only be undertaken when there is diagnostic uncertainty (eg, cardiac murmur with signs of congestive cardiac failure), age < 2 months, need for cohorting - i.e., influenza outbreaks.

Management

For initial treatment, refer to Table 26: Initial Management of Bronchiolitis.

Most children presenting in primary care have mild bronchiolitis. The main issues in management are parental reassurance and education about minimal handling and giving frequent feeds.

Clinical indications for admission to hospital are:

- difficulty feeding
- moderate to severe work of breathing
- increased respiratory rate. Assessment varies with the age of the child; for normal values according to age,
- oxygen saturation measured by pulse oximetry (SpO2) less than 94%.

Most children admitted to hospital require only symptomatic care with supplemental oxygen and minimal handling. Nasogastric feeds may be preferable to intravenous fluids if normal feeding is not possible. A small number of children with severe disease may require extra respiratory support with noninvasive ventilation (eg high-flow heated humidified nasal cannula therapy or continuous positive airway pressure [CPAP]) or invasive ventilation. These children require specialist care and referral to Paediatricians for further management is essential in severe disease or those with moderate disease with potential to deteriorate.

Table 26: Initial management of bronchiolitis based on severity of illness

The main treatment of bronchiolitis is supportive. This involves ensuring appropriate oxygenation and fluid intake, and minimal handling.

	Mild	Moderate	Severe
Likelihood of admission	Suitable for discharge	Likely admission. Discharge after a period of observation (must discuss with nearest paediatric facility)	Requires admission; consider appropriate children's facility / PICU
Observations *Vital signs (respiratory rate, heart reate, oxygen saturation, temperature)*	Adequate assessment in ED prior to discharge (minimum of two recorded measurements or every four hours)	1 - 2 hourly (not continuous) Once improving and not requiring oxygen for 2 hours discontinue oxygen saturation monitoring	Hourly with continuous cardiorespiratory (including oximetry) monitoring and close nurse observation.
Hydration / nutrition	Small frequent feeds	If not feeding adequately (less than 50% over 12 hours), administer NG hydration.	If not feeding adequately (<50% over 12 hours), or unable to feed, administer NG hydration
Oxygen saturation / oxygen reqirement	Nil requirement	If oxygen saturations fall below 90%, administer oxygen to maintain saturations ≥90% Once improving **and** not requiring oxygen for 2 hours, discontinue oxygen saturation monitoring	Administer oxygen to maintain saturations ≥90%

cont..

Respiratory support	N/A	Begin with NP oxygen (max 2L/min). If requiring higher flow, place on reservoir mask at 5-10 L/min and discuss with local paediatric team. HFNC to be used only if nasal prong oxygen has failed	Consider HFNC or CPAP. **Consider mechanical ventilation if in respiratory failure**
Disposition / escalation	Consider further medical review (in 24-48 hours) if early in the illness and any risk factors are present or if child develops increasing severity after discharge	Decision to admit should be supported by clinical assessment (including risk factors), social and geographical factors, and phase of illness	Consider ICU review/ admission to PICU if: • severity does not improve • persistent desaturations • significant or recurrent apnoea associated with desaturations • has risk factors
Parental education	• provide advice on the expected course of illness and danger signs. • frequent feeds and watch hydration status. • do **not** use OTC cough and cold medications. • encourage exclusive breastfeeding for at least 6 months. A parent information sheet is available from The Royal Children's Hospital (Melbourne): https://www.rch.org.au/kidsinfo/fact_sheets/Bronchiolitis/.		

ED = Emergency Department; CPAP = continuous positive ariways pressure; HFNC = high flow nasal cannula; NG = nasogastric tube; NP = nasal prong; OTC = over-the-counter; PICU = paediatric intensive care unit; SpO2 = oxygen saturation measured by pulse oximetry.

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Children with moderate disease have the potenital to deteriorate. Children with severe broncholitis should be refered for further management with the paediatricians at a divisional hospital.

Oxygen therapy

- oxygen therapy should be instituted when oxygen saturations are persistently <90%
- infants with bronchiolitis will have brief episodes of mild/moderate desaturations to levels <90%. these brief desaturations are not a reason to commence oxygen therapy
- oxygen should be discontinued when oxygen saturations are persistently ≥90%
- **once not requiring oxygen for 2 hours, discontinue oxygen saturation monitoring.** continue other observations 2 to 4 hourly and reinstate intermittent oxygen monitoring if deterioration occurs

Hydration/nutrition

- children are often more settled if comfort oral feeds are continued
- when non-oral hydration is required, nasogastric (NG) hydration is the route of choice
- if intravenous fluid is used, it should be isotonic with added glucose
- nasogastric or intravenous fluids should be commenced at two-thirds maintenance because of the potential for increased ADH secretion

Medication

- medications are not indicated in the treatment of bronchiolitis

The following medications are **not indicated** in the treatment of bronchiolitis. No evidence-based recommendations for use exist.

- beta 2 agonists - including infants with a personal or family history of atopy
- corticosteroids - nebulised, oral, intramuscular or intravenous
- adrenaline - nebulised, intramuscular or intravenous except in peri-arrest or arrest situation
- nebulised hypertonic saline (3%) - some evidence exist for use (decreased length of stay) but not currently recommended as standard therapy
- antibiotics – including azithromycin
- antivirals
- immunoglobulin
- montelukast

Antibiotics are **not indicated** in the outpatient management of acute bronchiolitis. However, they may be used in very ill hospitalised infants, especially if there is associated consolidation on chest X-ray. This is because secondary bacterial infection may complicate the illness, particularly in infants requiring ventilatory support. For antibiotic treatment in this setting, see the latest version of the *Antibiotic Guidelines*.

For further information on further management of severe bronchiolitis, refer to the Paediatric PICU CPG 2019.

Discharge Criteria

Consider discharge when:

- maintaining adequate oxygenation in room air
- feeding adequately
- apnoea-free for 48 hours (if the patient had apnoea).

Table 27: Respiratory rates in children

Age	Respiratory rates
Birth to 2mths	<60
2 months to <12mths	<50
12mths to 5years	<40
5years and above	<30

References: Integrated Management of Childhood Ilnesses, World Health Organization. Geneva, 2014 and Samuels, M. Wieteska, S (Eds). Advanced Paediatric Life Support: A Practical Approach to Emergencies; 6th Ed. 2016. John Wiley & Sons Ltd.

Croup

Introduction

Croup (acute laryngotracheobronchitis) is a common paediatric, viral respiratory tract illness. It typically occurs in children 6 months to 3 years of age and is chiefly caused by Parainfluenza viruses. The name acute laryngotracheobronchitis indicates that croup generally affects the larynx and trachea, although this illness may also extend to the bronchi.

Croup presents with a coryzal prodrome, hoarseness (or husky voice in those old enough to speak), inspiratory stridor, a harsh barking 'brassy' cough, and variable airway obstruction due to inflammatory oedema within the subglottis. It has a duration of 2 to 5 days; however, a postinfective cough may persist for many weeks. Parainfluenza viruses are the most common cause of croup, and antibiotics are not indicated.

Risk factors for severe croup

- pre-existing narrowing of upper airways
- subglottic stenosis (congenital or secondary to prolonged neonatal ventilation)
- down syndrome
- previous admissions with severe croup

Croup is uncommon in infants less than 6 months old, and rare in infants less than 3 months of age. Consider an alternative diagnosis for acute upper airway obstruction.

Clinical presentation

Children usually present with a sudden onset of barking cough, inspiratory stridor and increased work of breathing. They may have associated widespread wheeze, fever but no signs of toxicity. Refer to Table 28 for assessment of severity.

*Children with croup should have minimal examination. Do **not** examine the throat. Do not upset the child further.*

Investigations

Routine blood tests, imaging or nasopharyngeal swabs are not recommended.

Assessment of severity

Table 28: Severity assessment of croup

	Mild	Moderate	Severe
Behaviour	Normal	Some/intermittent irritability	Increasing irritability and/or lethargy
Stridor [Note 1]	Barking cough, stridor only when upset or active	Some stridor at rest	Stridor present at rest
Respiratory rate	Normal	Increased respiratory rate Tacheal tug Nasal flaring	Marked increased or decreased respiratory rate Tracheal tug Nasal flaring
Accessory muscle use	None or minimal	Moderate chest wall retraction	Marked chest wall retraction
Oxygen	No oxygen requirement	No oxygen requirement	**Hypoxaemia/ cyanosis is a late sign which indicates life-threatening croup.**

Note 1: The loudness of the stridor is not a good indicator to the severity of the obstruction.

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Restlessness, decreased level of consciousness, hypotonia, cyanosis and pallor are signs of life-threatening airway obstruction.

Investigations (nasopharyngeal aspirate, chest X-ray, blood tests) are **not** usually indicated and may cause the child distress and worsening symptoms.

Management

Initial treatment

There is good evidence to support the routine use of a single dose of corticosteroids in all children with croup, whether mild, moderate or severe. This has been shown to reduce hospital admission rates and prevent re-presentation.

*Hydrocortisone should **not** be used in the management of croup due to lack of evidence and short duration of action.*

Mild croup

Home treatment

Symptomatic care including antipyretics, mist, and oral fluids. Mist therapy (or humidified air) may provide a sense of comfort and reassurance to both the child and family, although studies have found only marginal improvement in croup scores. However, if the child is instead agitated by the mist, it should be discontinued.

Outpatient treatment

dexamethasone 0.15 mg/kg (maximum 16 mg), orally, as a single dose (dexamethasone injection (4 mg/mL) can be given orally; mixed with flavoured syrup)

OR

prednisone 1 mg/kg orally, with a repeated dose the following evening.

Observe for half an hour post steroid administration. If accessory muscle use, stridor at rest, or distress have not improved, **treat as for severe croup**.

Moderate croup

dexamethasone 0.15 to 0.6 mg/kg (maximum 16 mg) orally, as a single dose (dexamethasone injection (4 mg/mL) can be given orally; mixed with flavoured syrup)

OR

prednisolone 1 mg/kg orally, with a repeated dose the following evening.

PLUS

nebulised adrenaline 1:1000 (1 mg/mL) solution 0.5 mL/kg to maximum 5 mL (5 mg) undiluted by inhalation via nebuliser. Repeat dose of adrenaline after 30 minutes if no improvement.

If a second dose of nebulised adrenaline is required – **admit to ICU at a divisional hospital**.

Observe for a minimum of 4 hours after giving adrenaline. If accessory muscle use, stridor at rest, or distress have not improved, **treat as for severe croup**.

Severe croup

dexamethasone 0.6 mg/kg (maximum 16 mg) orally / IV/ IM, as a single dose (dexamethasone injection (4 mg/mL) can be given orally; mixed with flavoured syrup)

PLUS

nebulised adrenaline 1:1000 (1 mg/mL) solution 0.5mL/kg to maximum 5 mL (5 mg) undiluted.

If no improvement, repeat dose of adrenaline after 30 minutes. If a second dose of nebulised adrenaline is required – **admit to ICU at a divisional hospital**.

Consultation with anaesthesiologist or ENT surgeon may be warranted to arrange for intubation in a controlled setting.

Observe for a minimum of 4 hours after giving adrenaline.

If there is no response (eg ongoing stridor at rest) or deterioration occurs, give a further dose of adrenaline and immediately call the divisional paediatric consultant on-call.

Reconsider the diagnosis and consider differential diagnoses (eg bacterial tracheitis, inhaled foreign body, anaphylaxis).

For severe croup needing intubation:

- the child should be intubated if they have severe obstruction or a poor response to inhaled adrenaline. Do **not** wait until the child is exhausted or very severely obstructed.
- intubate the child with an oral endotracheal tube 0.5 - 1.0 mm smaller than the usual diameter. Usual size(mm) = (Age/4) + 4
- children should be extubated when there is a leak around the tube, or at 24 - 36 hours (>2 yr) or 36 - 48 hours (<2 yr). Some children may require intubation for longer.
- antibiotics are **not** usually indicated.

Additional notes

- inhalation of humidified air or steam provides no additional benefit, although may provide a sense of comfort and reassurance to both the child and family
- failure to respond may also be due rarely to bacterial tracheitis.

Divisional hospital admission

The decision to admit to hospital is made after initial treatment and observation. In children with severe croup, if there has been a good response to treatment with corticosteroids and/or nebulised adrenaline, observe the child for 4 hours or overnight before sending them home.

Consider consultation with CWMH / Lautoka / Labasa paediatric consultant when:

- No improvement following nebulised adrenaline
- More than 2 doses of nebulised adrenaline are required
- Children are requiring care above the level of comfort of the local hospital

The presence of ongoing stridor at rest (severe croup) necessitates admission to the ICU. Notify the ICU if the child has needed more than one dose of nebulised adrenaline in the emergency department.

Other key points of management

- Children with croup need minimal handling, this includes limited examination and nursing with parents
- Supplemental oxygen is not usually required, if needed consider severe airways obstruction
- Do not forcibly change a child's posture – they will adopt the posture that minimises airways obstruction
- IV cannulation should be deferred
- Avoid distressing the child further
- Children with cough only do not require treatment

Antibiotics have no role in uncomplicated croup as it has a viral aetiology – for moderate to severe croup where bacterial infection is suspected, give the first dose of appropriate antibiotic treatment and refer to a divisional hospital for follow up.

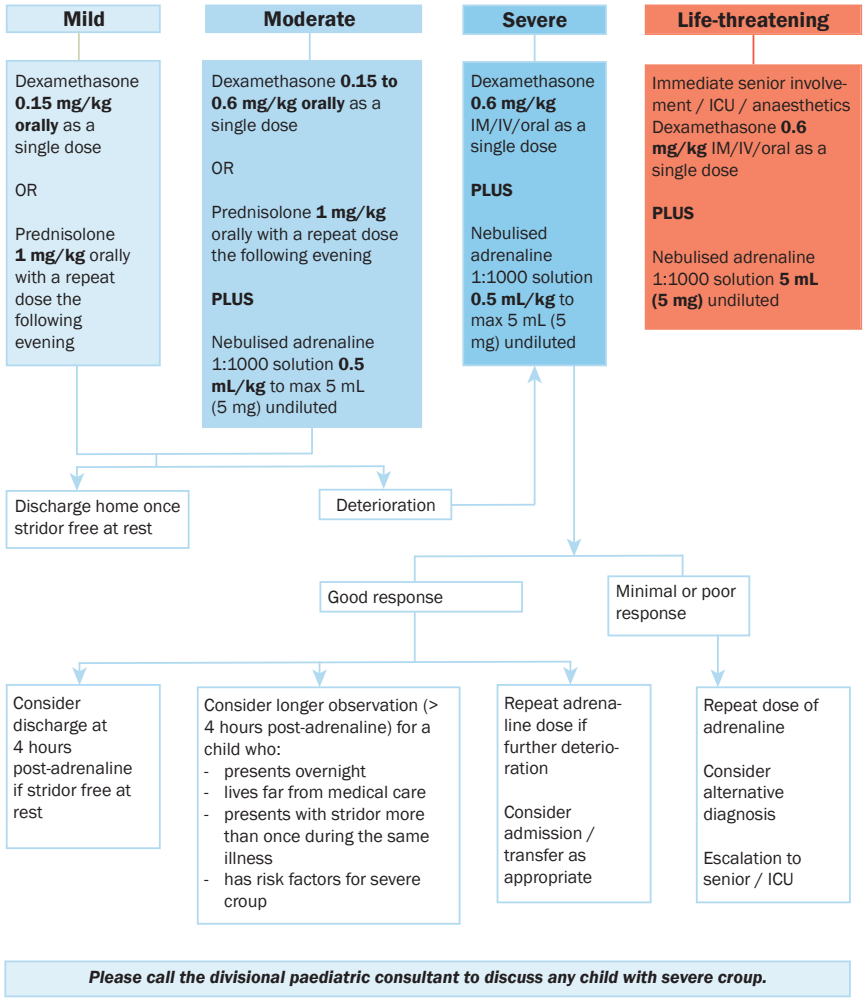
Do not use antitussives, such as codeine or pholcodeine, as they have no proven effect on the course or severity of croup and may increase sedation, thus interfering with assessment.

Discharge requirement

- Four hours post adrenaline nebs (if given) and/or half hour after oral steroid administration, and no stridor at rest. Arrange for follow-up within 24 hours.

Advise parents to seek help if stridor occurs at rest regardless of whether the patients have received steroids.

Figure 5: Croup management flowchart



Cystic fibrosis

Cystic fibrosis (CF) is an inherited autosomal recessive condition and is the most common cause of bronchiectasis and chronic suppurative lung disease in Caucasian children. However, it is extremely rare in non-Caucasian children. CF is a complex multisystem disease, which affects not only the lungs, gastrointestinal tract and pancreas, but also the liver, sinuses, sweat glands, bones and reproductive system.

Although previously considered primarily a paediatric disease, many patients with CF are now surviving to adulthood. Improvements in respiratory and nutritional management have increased median survival to over 35 years of age.

Progressive lung disease is the major cause of morbidity and mortality in CF This progressive lung damage involves a predisposition to recurrent bacterial infection and neutrophilic inflammation.

The aim of respiratory management is to limit the progression of lung injury. There are a number of treatment modalities, includingearly aggressive antibiotic therapy, chest physiotherapy and mucolytic agents, anti-inflammtory drugs, and new treatments correcting the abnormal gene product in CF. Any patient in Fiji with CF requires specialised care.

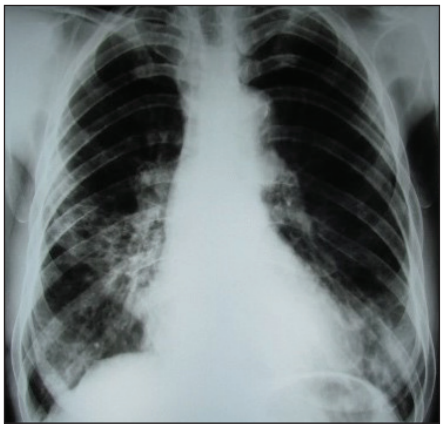
Bronchiectasis

Definition

Bronchiectasis is a disease characterised morphologically by the permanent dilatation of bronchi and bronchioles, and clinically by recurrent or persistent bronchial infection, cough and often sputum. The pathogenesis is related to chronic airway infection and inflammation resulting in airway damage.

Bronchiectasis may be classified under the chronic suppurative lung disease spectrum.

Image 3: Example of an X-ray image in bronchiectasis



Causes

Bronchiectasis may be localised to one lobe or segment or generalised in both lungs. Pneumonia, often in childhood, is the most commonly recognised cause of bronchiectasis. However, in one series, no definite cause could be established in 51% of patients. When focal disease is present, the cause may be an intraluminal obstruction (eg an unsuspected foreign body or endobronchial tumour), or extrinsic compression of the airway by enlarged lymph nodes.

Bronchiectasis can be associated with many other conditions, including inherited and acquired immune deficiencies, and various autoimmune diseases.

Some of the following causes may be important to consider in Fiji:

- inherited immune deficiencies in children and adults, particularly deficiencies of immunoglobulin G (IgG) and immunoglobulin M (IgM)
- autoimmune diseases including rheumatoid arthritis and inflammatory bowel disease
- acquired immunodeficiencies and recurrent pneumonia
- recurrent sepsis in patients with sinus disease and suppurative otitis media
- recurrent aspiration where there are neurological or anatomical disorders affecting oropharyngeal muscle coordination and swallowing
- fibrotic lung diseases such as idiopathic pulmonary fibrosis causing traction bronchiectasis
- Mycobacterial infections

Bronchiectasis may coexist with asthma or chronic obstructive pulmonary disease (COPD).

Clinical features and diagnosis

Most adult patients with bronchiectasis have a chronic cough with sputum production. Children may present with a persistent wet cough, as they are unable to expectorate sputum. The sputum is usually purulent and may be intermittently bloodstained. Consider bronchiectasis in patients who have persistent symptoms not responding to standard treatment and/or in whom a Gram-negative organism, such as Pseudomonas aeruginosa, is found on sputum culture. Patients may present with life-threatening haemoptysis requiring urgent hospitalisation.

Wheezing and shortness of breath are common during exacerbations, and pleurisy may occur if distal pneumonia is present. Crackles and wheeze are commonly heard, although chest examination in children may be normal. A small proportion of patients (less than 5%) have clubbing of the fingers.

High-resolution computed tomography (HRCT) scan of the chest is the preferred test to confirm the diagnosis of bronchiectasis because it allows clear visualisation of the severity and distribution of the disease. Pulmonary function testing often reveals associated airflow limitation. Diagnostic testing for some of the specific conditions associated with bronchiectasis is mandatory in children and may be appropriate in adults, depending on the clinical setting and/or radiological appearances.

Patient history and physical examination should direct investigations. If there is no obvious clue, basic investigations should be done (full blood count (FBC), erythrocyte sedimentation rate (ESR), biochemistry (creatinine, urea and liver function tests), and chest X-ray), and could include immunoglobulin concentrations (including IgG subtypes) and an autoimmune screen (rheumatoid factor [RF] and antinuclear antibodies [ANA]).

Management

Overview

The basic aims in bronchiectasis management are to keep the airways as free of secretions as possible, the background microbiological load low and the number of infective exacerbations to a minimum. This involves treating all acute respiratory tract infections and managing any underlying conditions. Rarely, surgically removing a severely damaged section of lung may be helpful.

Baseline management is outlined below. **The following are suggested indications for seeking specialist opinion:**

- rapid progression of disease or symptoms
- disease requiring hospitalisation
- severe respiratory symptoms or lack of response to current treatment
- frequent need for antibiotics, such as more than 3 to 4 courses of antibiotics within 12 months
- resistant or unusual organisms isolated in sputum
- haemoptysis
- clinical deterioration indicated by
  - inability to maintain weight
  - declining lung function.

General measures

Keeping the airways as free of secretions as possible is an important part of the management of bronchiectasis. Modern methods of airway clearance, including physical techniques (various breathing and coughing techniques) and drug treatment, allow greater patient independence.

Tapping with postural drainage is no longer the preferred method of airway clearance. Where possible, refer patients to an experienced respiratory physiotherapist to develop an individualised sputum-clearing program.

Nebulised agents that may assist with airway clearance include bronchodilators and normal and hypertonic saline (sodium chloride solution). Treat any reversible component of airflow limitation with bronchodilators and inhaled corticosteroids.

It is important for patients to maintain weight, muscle strength and mass through good nutrition and exercise. Exercise may be useful in promoting respiratory secretion clearance.

Lung function testing should be performed initially to assess lung function. Spirometry should be done when clinically indicated to monitor airflow limitation.

Immunisation with pneumococcal vaccine and annual influenza vaccine is recommended (not on Fiji EML at time of publishing).

Antibiotic therapy

Acute infective exacerbations of bronchiectasis should be treated for 10 to 14 days with oral amoxycillin or doxycycline. Proven *Pseudomonas aeruginosa* infections can be treated with ciprofloxacin for 14 days. For details, including intravenous treatment for severe exacerbations, consult the latest version of the *Fiji Antibiotic Guidelines*.

Long-term treatment

Patients with bronchiectasis often have chronically purulent sputum. If cultured, it grows organisms such as *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Moraxella catarrhalis, Pseudomonas aeruginosa* and *Staphylococcus aureus*. The isolation of *Aspergillus* is usually not clinically significant and does not need to be treated. The presence of *P. aeruginosa* in the airways of these patients is generally associated with more severe disease. Sputum cultures should be taken initially and when clinically indicated, to determine the usual colonising organism. If a patient is clinically stable, it is not appropriate to treat colonising organisms as this will promote the emergence of antibiotic resistance.

In a clinically stable patient, it is not appropriate to treat colonising organisms.

If an acute exacerbation occurs, a new sputum sample should be collected for culture and susceptibility testing because the infective agent may not be the usual colonising organism. Treatment against the last organism isolated can be started before results are available. If possible, avoid long-term use of antibiotics for bronchiectasis because it is likely to promote the development of antibiotic resistance.

Management of haemorrhage

In adults with bronchiectasis, minor haemoptysis may be an indication of infection and should be treated with antibiotics but does not necessarily require further investigation. If possible, stop any drugs that may promote haemorrhage (eg nonsteroidal anti-inflammatory drugs [NSAIDs], anticoagulants).

Massive haemoptysis (more than 250 mL in 24 hours) can occur with bronchiectasis. If this happens, immediately send the patient to a hospital where appropriate specialist services are available. Investigations are generally aimed at localising the site of haemorrhage and may include computed tomography (CT) scan of the chest with angiography and bronchoscopy. Treatment options include lobectomy.

For the management of cor pulmonale, see ‘COPD: Pulmonary hypertension and cor pulmonale’ page 103, and respiratory failure, see ‘COPD: ventilatory support’ page 110 and ‘Non-invasive ventilation’ page 167.

Bronchiectasis in children

Bronchiectasis not associated with cystic fibrosis may occur in children. Appropriate investigation of potential causes and intensive treatment (including airway clearance, preventive immunisations, antibiotic therapy, and optimisation of nutrition and growth) are key to reducing long-term lung damage and maintaining lung function. Children should be managed by a specialist paediatric clinic.

Pleural disease

Pleuritic pain

Pleuritic pain has many causes. Common causes are:

- inflammation of the pleura caused by viral or bacterial pneumonia
- pulmonary infarction caused by pulmonary embolism
- pneumothorax
- connective tissue disease
- trauma

An accurate diagnosis is important. Provide sufficient analgesia during investigations to allow adequate respiratory movements and so decrease the risk of atelectasis and pneumonia.

Nonsteroidal anti-inflammatory drugs (NSAIDs) may be appropriate initially; however, opioids may be needed. The cough suppressant and respiratory depressant actions of opioids should be considered before use. If opioids are required for patients with chronic obstructive pulmonary disease or patients thought to be hypoxaemic, admission to hospital for monitoring is advised.

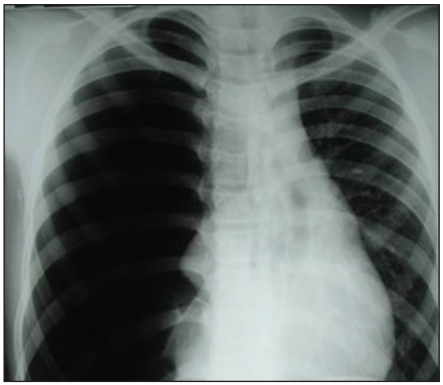
Pneumothorax

Pneumothorax is the presence of air between the parietal and visceral pleura, and can be classified as spontaneous, traumatic or iatrogenic.

Spontaneous pneumothoraces are subdivided into:

- primary—where there is no evidence of underlying lung disease
- secondary—where lung disease is present, most commonly chronic obstructive pulmonary disease (COPD) but also asthma, interstitial lung disease, cystic fibrosis or HIV-associated infection.

Image 4: An example of an X-ray in pneumothorax



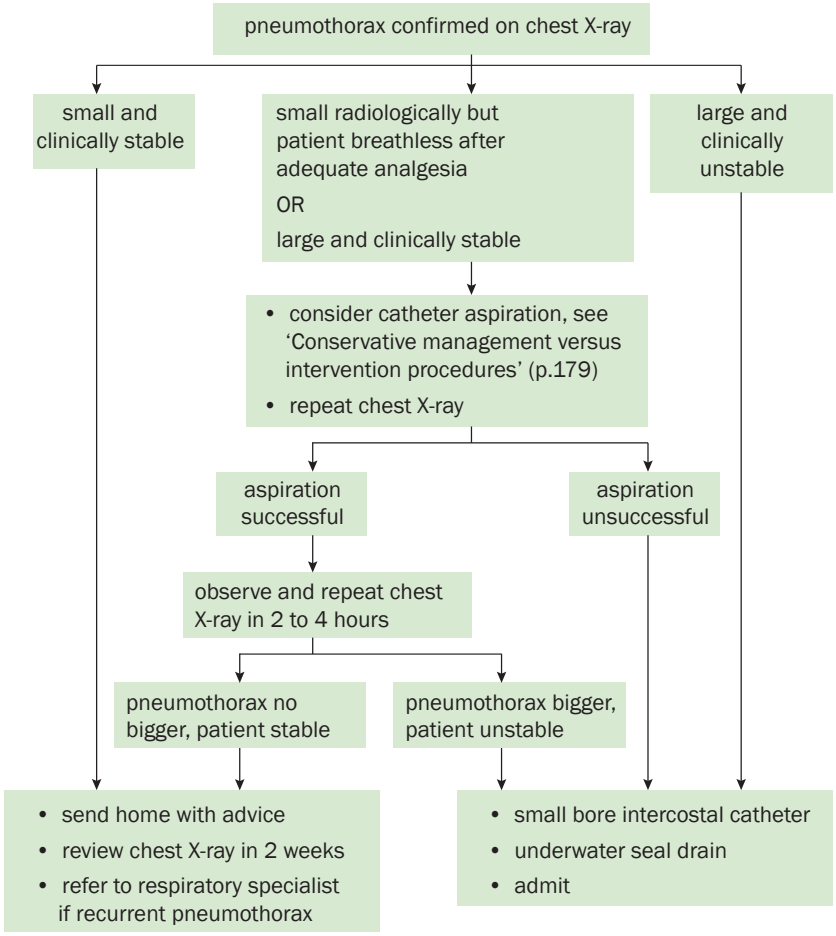
Spontaneous pneumothorax usually presents with sudden onset of pleuritic chest pain and breathlessness, and is diagnosed primarily on history and chest X-ray, preferably taken with the patient erect. Pneumothoraces may be difficult to see on plain films taken in the supine position, and computed tomography (CT) scan may be required.

Specific chest signs depend on the size of the pneumothorax and may be difficult to detect. In a large pneumothorax, physical signs include absent breath sounds, tachypnoea, decreased chest wall movement, hyperresonance to percussion, decreased vocal resonance and tracheal deviation to the opposite side.

For a summary of the approach to management of spontaneous pneumothorax, see below.

All patients with pneumothorax should be discussed with a specialist in divisional hospital.

Figure 6: Management of spontaneous pneumothorax



Decompensated pneumothorax

Decompensated pneumothorax (also known as tension pneumothorax) is a medical emergency and requires urgent decompression. The rapid development of a large pneumothorax, particularly if there is underlying lung disease or trauma, may result in severe breathlessness, hypoxaemia, hypotension and cardiovascular collapse.

The diagnosis is clinical not radiological. Mediastinal shift is seen in many large pneumothoraces and does not of itself indicate a need for decompression.

The concept that the effects of a decompensated pneumothorax relate to supra-atmospheric intrapleural pressure has been questioned, and the term decompensated pneumothorax is preferred to tension pneumothorax. However, patients on positive pressure ventilation, including continuous positive airway pressure (CPAP), may develop intrathoracic pressures exceeding atmospheric pressure and deteriorate rapidly.

Decompensated pneumothorax is extremely rare in primary spontaneous pneumothorax.

Management

Primary spontaneous pneumothorax

Conservative management versus intervention procedures

Primary spontaneous pneumothorax is usually a nuisance rather than a dangerous condition. Symptoms commonly resolve within 24 to 48 hours without treatment.

Conservative management of even large, primary spontaneous pneumothoraces was usual in the past with excellent results, and is increasingly being used. Drainage of pneumothorax is often painful and carries risks (including damage to internal organs, bleeding and infection).

If an intervention procedure for pneumothorax is performed, there is good evidence that aspiration is preferable to insertion of an intercostal drain. Earlier and more vigorous intervention is associated with increased complications.

Existing guidelines advise that patients with small (less than 2 cm) pneumothoraces who are stable may be observed. Patients can be considered clinically stable if they:

- are not short of breath
- are able to speak in sentences
- have a respiratory rate less than 24 breaths/minute
- have a pulse rate between 60 and 120 beats/minute
- have a blood pressure that is normal for them.

The objectives of treatment are to relieve symptoms, minimise hospital admission and prevent recurrence.

Adequate analgesia is important and often substantially improves breathlessness, which may be largely related to the pain of breathing.

If the patient is in hospital, oxygen may be given not only to maintain oxygenation but also to increase the rate of resorption of the intrapleural air. Use:

oxygen 10 L/minute via face mask.

Persistent air leak

A persistent air leak (bronchopleural fistula) develops in about one-third of cases of pneumothorax treated with intercostal drains. It is more common in secondary spontaneous pneumothorax. One study has shown that all persistent air leaks in primary pneumothorax resolved in 15 days and up to 80% of persistent air leaks in secondary spontaneous pneumothorax resolved in 14 days. Early surgical intervention is sometimes advised but there is no evidence to support this.

Recurrence

The risk of a recurrence of spontaneous pneumothorax is estimated to be 30 to 50%; continuing smokers have higher risk. If there has been one recurrence on the same side, the risk of further recurrences rises sharply and referral for pleurodesis is recommended. Pleurodesis techniques include medical thoracoscopy with talc insufflation, surgical video-assisted thoracoscopy with pleural abrasion, and injection of talc slurry through an intercostal catheter.

Secondary spontaneous pneumothorax

Patients with secondary spontaneous pneumothorax generally require early active intervention and hospitalisation for observation. Existing guidelines recommend intercostal drainage except for patients who are not breathless and have a very small or apical pneumothorax. Simple aspiration is less likely to be successful and is only recommended as initial treatment in small pneumothoraces in patients with minimal symptoms and who are younger than 50 years.

Iatrogenic pneumothorax

Iatrogenic pneumothorax may occur following pleural aspiration, or transbronchial or percutaneous lung biopsy. They are usually small and resolve spontaneously, but aspiration may be considered.

Pneumothorax decompression methods

Urgent needle decompression

If the patient is *critically* ill and impending collapse, do not delay decompression while arranging for sterile procedures and local anaesthetic (although these are desirable).

For urgent needle decompression of a pneumothorax:

- insert a cannula above the third rib in the mid-clavicular line
- remove the needle from the cannula
- a gush of air confirms the diagnosis
- once complete, insert a thoracostomy tube expeditiously.

Catheter aspiration

For catheter aspiration of a pneumothorax (sometimes termed thoracocentesis), see below.

Box 7: Catheter aspiration of a pneumothorax

For catheter aspiration of a pneumothorax:

- Use a small-bore catheter, such as a venous catheter with a 3-way valve or a pigtail catheter, or a catheter such as a single-lumen central line.
- Using an aseptic technique, identify the third rib in the mid-clavicular line.
- At this surface landmark, infiltrate local anaesthetic (5 to 10 mL of 1% lignocaine) subcutaneously and deeper until reaching the pleural space; this must be confirmed by aspiration of air into the syringe.
- Insert the catheter above the third rib in the mid-clavicular line, unless the pneumothorax is elsewhere.
- Aspirate until no more air is returned.
- Leave the catheter in situ and immediately repeat the chest X-ray.
- Repeat the chest X-ray again in 2 to 4 hours
  - If the pneumothorax has not reaccumulated, remove the catheter
  - If the pneumothorax has reaccumulated, connect the catheter to a continuous drainage underwater seal or Heimlich valve
- If there is no reaccumulation, discharge the patient with advice to return if symptoms recur, or every 2 weeks until the pneumothorax has resolved.

Intercostal tube drainage

Intercostal tube drainage (tube thoracostomy) is indicated in the following circumstances:

- if simple aspiration fails in primary spontaneous pneumothorax
- in secondary spontaneous pneumothorax unless the patient is asymptomatic and the pneumothorax is very small
- in most cases of traumatic pneumothorax
- in decompensated (tension) pneumothorax.

For simple pneumothorax, preferably connect a small-bore catheter (10 to 14 Fr gauge) either to a Heimlich valve or to an underwater seal. There is no evidence that insertion using a Seldinger technique reduces complications. Suction is not indicated. Large-bore catheters are used in traumatic pneumothorax to allow for drainage of blood.

Intercostal tube drainage is a specialised procedure; for further information see British Thoracic Society guidelines

Pleural effusion

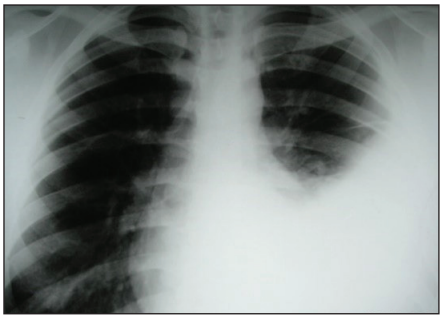
Introduction

Pleural effusion is buildup of fluid in the pleural space. Pleural fluid analysis is important as the appropriate management of pleural effusion depends on an accurate diagnosis. Effusions can be either transudates or exudates. Most transudates occur in clinically obvious situations of heart failure, liver cirrhosis, nephrotic syndrome, or in patients receiving peritoneal dialysis. There are many causes of exudative pleural effusions including malignancy, tuberculosis and parapneumonic effusion.

Use direct ultrasound guidance to sample pleural fluid by aspiration.

Use direct ultrasound guidance to sample pleural fluid by aspiration. This can be safely undertaken at the bedside, sampling with a 21 G (green) needle and a syringe. Obtain as large a sample as possible to analyse for biochemistry (which may include pH, lactate dehydrogenase [LDH], protein and glucose), cytology, microbiology (cell count and differential) and culture (including for mycobacteria), and/or molecular test for tuberculosis (Xpert MTB-RIF). Note the gross appearance of the fluid. If clinical suspicion of tuberculosis is high, or if the fluid is clearly blood stained (suggesting malignancy), consider a pleural biopsy. If the pleural fluid is frankly pus or blood avoid pleural biopsy. If the pleural fluid is serous then consider biopsy to look for TB or malignancy as clinically correlated.

Image 5: An example of an X-ray image in pleural effusion



Supportive therapy for pleural effusion includes:

- pain relief
- oxygen
- IV fluids

All patients with pleural effusion should be discussed with a specialist at the nearest divisional hospital.

Management of parapneumonic effusion and empyema

Introduction

Pleural effusion complicates up to 50% of cases of pneumonia; it is then called a parapneumonic effusion. This is sterile initially, but if not detected and managed appropriately may develop into a thoracic empyema. Any clinically significant parapneumonic effusion confirmed on chest X-ray requires diagnostic sampling and culture of the fluid sample.

Empyemas, as with any collection of pus, always require adequate drainage and antibiotic therapy.

Antibiotic therapy should be directed by cultures wherever possible, but many cases are culture negative because of previous antibiotic use. In these cases, therapy should be directed at the most likely organisms, which in pleural infection accompanying community acquired pneumonia are Strep pneumoniae or a member of the Strep "milleri" group. Recommended empirical treatment is oral amoxycillin plus clavulanic acid. Severely ill patients should be treated as for severe sepsis.

Empirical treatment of pleural infection secondary to hospital acquired pneumonia should be with the same antibiotics as for the pneumonia.

For details of the antibiotic therapy of empyema consult the latest version of the *Antibiotic Guidelines*.

Drainage

Indications for drainage of parapneumonic effusion are:

- continued fever and sepsis despite adequate antibiotic therapy
- large size (more than one-third of the hemithorax)
- loculated pleural effusions
- evidence of continued pleural infection, such as
  - frankly purulent or turbid fluid on sampling
  - presence of bacteria on Gram stain or culture of pleural fluid
  - pleural fluid pH less than 7.2
  - pleural fluid LDH concentration more than 1000 units/L.

Intrapleural enzyme therapy

Recombinant human DNase (dornase alfa) has been shown to reduce intrapleural pus viscosity, and DNase combined with a fibrinolytic (recombinant tissue plasminogen activator [alteplase]), significantly reduces length of hospital stay and the need for surgery and improves radiographic appearance. However, neither of these agents is available on the Fiji EML. Alteplase may be available through the Fiji FMP, contact FMP Pharmacist at FPBSC.

Intrapleural fibrinolytic enzymes were previously recommended as monotherapy to treat empyema. However, a randomised multicentre study of intrapleural administration of streptokinase has shown that its use does not improve mortality, need for surgery, final radiographic appearance or length of hospital stay, and fibrinolytics alone are no longer recommended.

Surgery

For patients in whom sepsis continues despite adequate antibiotic therapy and tube drainage (including the placement of additional catheters under imaging into the other loculations if necessary), consult a thoracic surgeon to consider thoracoscopy, including video-assisted thoracoscopic surgery, or thoracotomy and open drainage.

Persisting chest X-ray abnormalities in a well patient are not an indication for surgery and are likely to improve over time.

Management of malignant pleural effusions

The presence of pleural effusion in malignancy is an adverse prognostic factor. Symptomatic malignant pleural effusions may require drainage. Reaccumulation of fluid after drainage is very common. More definitive treatment with pleurodesis depends on individual patient circumstances and preferences.

Pleurodesis can be performed as dry talc poudrage at thoracoscopy, or by instillation of a sclerosant (eg talc slurry) through an intercostal catheter. The latter entails drainage of the fluid from the pleural space, then instillation of a talc slurry or other sclerosing agent under local anaesthesia. The tube remains in place until the daily fluid drainage is less than 150 mL. Graded talc with a large particle size reduces the risk of acquired respiratory distress syndrome (ARDS), which has been reported with ungraded talc.

Instillation of talc and other sclerosing agents into the pleural space can cause considerable pain, although this is less common with malignant effusions. As well as lignocaine before instillation of the sclerosant, parenteral morphine is required for premedication and for treatment of pain following the procedure.

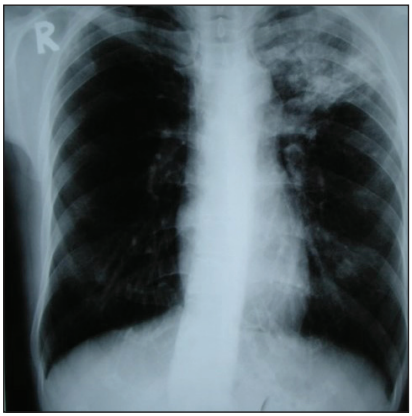
Some centres use sclerosants other than talc (including minocycline, bleomycin and mustine hydrochloride) with limited success.

An alternative to pleurodesis in cases where the lung fails to re-expand (trapped lung) or in the case of patient preference, is the placement of a permanent indwelling tunnelled pleural catheter.

Tuberculous pleural effusion

Tuberculous infection is common in Fiji. Pleural effusion is not an uncommon presentation of extra pulmonary Tuberculosis. It should always be considered in any patient with unexplained unilateral pleural effusion. Please refer to the *Fiji Tuberculosis Manual* for evaluation and management.

Image 6: An example of an X-ray image in a patient with a sputum positive for *Mycobacterium tuberculosis*

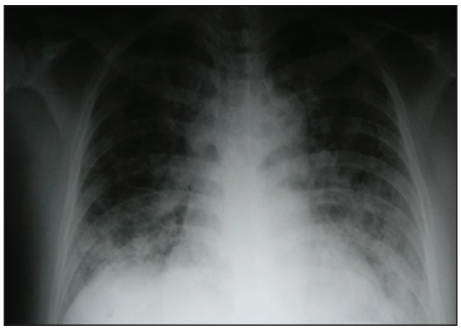


Interstitial lung disease

Introduction

The interstitial lung diseases (ILDs) are a diverse group of largely unrelated conditions. They are classified together because of a shared pathology centred on the interstitial compartment or parenchyma of the lung. They share varying degrees of inflammation and/or fibrosis of the interstitium or lung parenchyma. The terms ILD and lung fibrosis are used interchangeably.

Image 7: an example of an X-ray image in interstitual lung disease



Many of the ILDs are of unknown aetiology; the commonest are idiopathic pulmonary fibrosis (IPF) in older people and pulmonary sarcoidosis in younger adults. Additionally, there are a number of environmental exposures and other systemic diseases that can result in and be accompanied by lung fibrosis. A classification of the major interstitial lung diseases is shown in Box 7: Classification of major interstitial lun diseases.

Patterns of disease behaviour, prognosis and response to therapies vary between the different ILDs. The rate of clinical progression from initial symptoms to end-stage disease can extend from a few months to many years. A classification of the ILDs based on expected clinical course has recently been suggested and may be useful in deciding which patients will benefit from drug treatment, see the table below.

Box 8: Classification of major interstitial lung diseases

Idiopathic interstitial pneumonias

- idiopathic pulmonary fibrosis
- nonspecific interstitial pneumonia
- desquamative interstitial pneumonia
- respiratory bronchiolitis–interstitial lung disease
- cryptogenic organising pneumonia

Multisystem disorders

- connective tissue disease
- sarcoidosis
- inflammatory bowel disease

Environmental exposures

- hypersensitivity pneumonitis
- pneumoconiosis
- drug-induced interstitial lung disease
- radiation-induced interstitial lung disease

Genetic

- familial idiopathic pulmonary fibrosis
- short telomeres

Miscellaneous

- histiocytosis
- lymphangioleiomyomatosis
- pulmonary alveolar proteinosis
- eosinophilic pneumonia

Table 29: Clinical course of some interstitial lung diseases

Examples of ILD	Expected clinical course
RB–ILD	reversible and self-limited disease
hypersensitivity pneumonitis	
cellular NSIP	reversible disease with risk of progression
histiocytosis	
pulmonary sarcoidosis	
fibrotic NSIP	may be stable with residual disease, or
	may be progressive irreversible disease with potential for some stabilisation
IPF	progressive irreversible disease despite therapy

ILD = interstitial lung disease; IPF = idiopathic pulmonary fibrosis; NSIP = nonspecific interstitial pneumonia; RB–ILD = respiratory bronchiolitis–interstitial lung disease

Presentation

Patients with interstitial lung disease (ILD) typically present with progressive dyspnoea, dry cough and/or incidental findings of crackles on auscultation. The latter are often associated with an initial misdiagnosis of pulmonary oedema. Advanced disease presents with tachypnoea, tachycardia and low oxygen saturation measured by pulse oximetry (SpO2). Clubbing may be present. Early in the onset of ILD, there may be clinical signs (such as crackles) similar to those seen with pneumonia.

For all suspected interstitial lung disease patients, refer to a specialist at your nearest divisional hospital.

Investigations

If interstitial lung disease (ILD) is suspected, consider investigations to exclude other causes of dyspnoea and crackles. For example, full blood count (FBC), chest X-ray and echocardiogram to exclude anaemia, pneumonia or heart failure.

Some blood tests can help to determine the underlying cause of lung fibrosis, for example full blood count (FBC) with eosinophil count, antinuclear antibodies (ANA), double-stranded DNA (dsDNA), serum angiotensin converting enzyme (sACE), serum calcium, rheumatoid factor (RF), cytoplasmic antineutrophil cytoplasmic antibiotics (cANCA).

Patients with ILD develop a restrictive defect in pulmonary function. This is characterised by reduced forced vital capacity (FVC) and forced expiratory volume in

1 second (FEV_1). Changes in pulmonary function tests over time are used to follow disease progression - a 10% decline in FVC is defined as being significant.

Lung fibrosis is typically first suspected on chest X-ray, although a high-resolution computed tomography (HRCT) scan is required to clarify the diagnosis.

HRCT is the pivotal diagnostic test in ILD. The pattern of distribution can help to distinguish the different ILDs. Peripheral ILDs include eosinophilic pneumonia and cryptogenic organising pneumonia. Upper zone predominant diseases include granulomatous disorders (sarcoidosis, chronic hypersensitivity pneumonitis), while lower zone distribution diseases include IPF and nonspecific interstitial pneumonitis.

Bronchoscopy is indicated for patients presenting with haemoptysis or acute onset ILD with rapidly progressing symptoms and radiographic infiltrates. A transbronchial biopsy is appropriate in suspected granulomatous ILD and is useful to culture if an infective (eg tuberculosis) or malignant (eg lymphangitis) cause is suspected.

Specific adult interstitial lung diseases

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF) (previously termed cryptogenic fibrosing alveolitis) is typically a disease of older people, with a median age of onset of 67 years. It is slightly more common in males and smokers. The cause is unknown. A family history will be present in 10% of patients.

The diagnosis of IPF requires:

- exclusion of other known causes of interstitial lung disease (ILD), eg environmental exposures, drug toxicity, connective tissue disease
- typical high-resolution computed tomography (HRCT) pattern if patients are not undergoing surgical lung biopsy.

HRCT has a high specificity for diagnosing IPF. Surgical lung biopsy is not required in most cases; it is reserved for patients with either an atypical history or nonclassical radiology.

The natural history of IPF is variable and unpredictable. It is characterised by a gradual worsening of lung function over years and in most cases is fatal 3 to 5 years from diagnosis. Disease progression is demonstrated by increasing respiratory symptoms, deteriorating pulmonary function tests and worsening appearance of fibrosis on HRCT.

While commonly prescribed, there is no evidence to support the use of prolonged duration corticosteroid monotherapy although acute exacerbations can be treated with short courses of prednisone.

Immunosuppressants and long-term corticosteroids should only be commenced by a specialist at a divisional hospital.

Nonspecific interstitial pneumonia

Nonspecific interstitial pneumonia is commonly seen in association with connective tissue diseases (eg scleroderma, rheumatoid arthritis, polymyositis) but can be idiopathic. The clinical course is highly heterogeneous, with an accelerated course seen in patients with greater amounts of fibrosis. Overall, survival is significantly better than that seen with IPF.

Treatment is reserved for patients with severe or progressive disease. For patients with connective tissue disease, therapy that is already prescribed for the underlying disease (eg cyclophosphamide, azathioprine, methotrexate) may positively influence the natural history of the ILD. Otherwise, first-line therapy includes corticosteroids given for 3 months with or without corticosteroid-sparing drugs, such as cyclophosphamide or azathioprine. Total duration and intensity of therapy is tailored to clinical, physiological and radiological response.

Smoking-related interstitial lung disease

Respiratory bronchiolitis–interstitial lung disease, Langerhan cell histiocytosis and desquamative interstitial pneumonia have characteristic radiological and histological patterns. All three are strongly associated with smoking, but complete resolution can be seen if patients stop smoking. Refractory cases can be treated with corticosteroids.

Pulmonary sarcoidosis

Sarcoidosis is a chronic, multiorgan granulomatous disorder of unknown aetiology. It may present as asymptomatic hilar lymphadenopathy, which may be found incidentally, and has a self-limiting disease course. Mediastinal lymphadenopathy is seen in 90% of cases. Symptoms related to lung disease include dyspnoea on exertion, and cough.

Oral corticosteroids are indicated in dyspnoeic patients with pulmonary infiltrates and worsening pulmonary function tests. The dose used would be:

> prednisolone 0.5 mg/kg (up to 50 mg) orally, daily for 4 weeks, with subsequent duration and intensity of therapy tailored to clinical, physiological and radiological response.

Other therapy may be primarily targeted to extrathoracic disease.

Hypersensitivity pneumonitis

Hypersensitivity pneumonitis (previously termed extrinsic allergic alveolitis) is a granulomatous condition whose chronic form can be associated with lung fibrosis. Common forms of chronic hypersensitivity pneumonitis include bird fancier's lung (exposure to proteins found in feathers or droppings) and hot tub lung (exposure to Mycobacterium avium complex). The key to management is avoidance of the inciting environmental exposure because continued antigen exposure is associated with chronic disease and progression to lung fibrosis. Corticosteroids are recommended in acute, severe and progressive disease. The dose used would be:

> prednisolone 0.5 mg/kg (up to 50 mg) orally, daily for 4 weeks, with subsequent duration and intensity of therapy tailored to clinical, physiological and radiological response.

Childhood interstitial lung disease

Childhood interstitial lung disease is rare. As in adult ILD, some forms of ChILD respond to corticosteroids. The response to therapy and overall prognosis is better than in most adult forms of ILD. The majority of children recover and most can lead normal lives. However, some subgroups of ChILD, particularly those with genetic abnormalities of surfactant function, have a poor prognosis.

Any child with suspected ChILD should be referred to a paediatrician at a divisional hospital.

Sleep-disordered breathing

Sleep-disordered breathing in adults

Introduction

Sleep-disordered breathing refers to a variety of problems with breathing during sleep, including:

- obstructive sleep apnoea (OSA)
- OSA coexisting with chronic lung disorder
- central sleep apnoea
- obesity hypoventilation

Obstructive sleep apnoea

Overview

Obstructive sleep apnoea is the most common form of sleep-disordered breathing. The obstructive sleep apnoea syndrome (also called obstructive sleep apnoea–hypopnoea syndrome) refers to the presence of apnoeas and hypopnoeas during sleep together with daytime dysfunction, predominantly excessive daytime sleepiness.

The typical patient with sleep apnoea is overweight, middle-aged, male and often has a short thick neck, and a history of alcohol and tobacco consumption. However, women and thin young people can also develop sleep apnoea, especially those with a narrow anteroposterior diameter to their pharynx (which may occur in people of Asian origin) or those with a high arched palate (which may occur in Marfan syndrome).

Increasing evidence suggests that untreated obstructive sleep apnoea is associated with:

- hypertension
- arrhythmias such as paroxysmal atrial fibrillation
- cardiovascular mortality
- cerebrovascular mortality
- motor vehicle accidents, especially single-vehicle accidents at night
- pregnancy-related hypertension and pre-eclampsia.

In view of the potential for impaired driving, patients with severe obstructive sleep apnoea are advised to stop driving vehicles and operating machinery until their condition has been adequately stabilised on treatment. However, it is not possible to accurately predict individual driving risk, even for patients with severe sleep apnoea, as other factors are also important, such as acute sleep deprivation, circadian

misalignment (eg shift work and jet lag), narcolepsy, alcohol, comorbidities and other drugs.

Patients may benefit from general advice on good sleep practices.

Clinical assessment

The symptoms and possible associated diseases suggestive of obstructive sleep apnoea are listed in Box 9.

The Berlin questionnaire is a validated checklist that can be used to identify patients who may have sleep apnoea.

Patients do not always recognise significant daytime dysfunction despite severe disturbance of their nocturnal breathing; their partner may give a better indication of their daytime sleepiness.

Restless legs syndrome is a separate entity, but it may coexist with obstructive sleep apnoea and worsen sleep independently of the sleep-disordered breathing.

Box 9: History-taking in suspected obstructive sleep apnoea

Check for the following **symptoms** (with both the patient and their partner):

- snoring—frequency, loudness, and whether the partner needs to leave the bedroom
- waking through the night with a sensation of choking
- nocturnal interruption to breathing recognised by the partner, termed 'witnessed apnoeas'
- nocturia
- nocturnal sweating
- daytime sleepiness and irritability
- poor concentration or attention span
- morning headache
- gastro-oesophageal reflux
- erectile dysfunction

Ask about possible coexisting disease:

- hypertension, especially in younger patients
- cardiovascular disease, especially atrial fibrillation and heart failure
- cerebrovascular disease
- diabetes mellitus
- thyroid disease
- family history of sleep apnoea or continuous positive airway pressure (CPAP) use.

Diagnostic tests

The standard diagnostic test for obstructive sleep apnoea is the overnight in-laboratory polysomnogram. This allows detailed monitoring of body position and sleep stage, the two most important variables influencing sleep-disordered breathing.

At the time of publishing, these diagnostic tests were only available in the private sector in Fiji.

Simplified home-based assessments vary greatly in complexity from simple oximetry screening to almost a full polysomnogram. Home-based tests may be appropriate in some settings as a screening tool for patients with high pre-test probability of obstructive sleep apnoea (eg sleepy obese patients who snore and take antihypertensive drugs). Home-based tests can be useful to rule in obstructive sleep apnoea in these patients, but not to rule out obstructive sleep apnoea as the tests have high specificity and low sensitivity.

Treatment

General measures

Many patients with obstructive sleep apnoea may need treatment with continuous positive airway pressure (CPAP). However, address the following measures where possible, especially in mild disease with no daytime sleepiness, no impairment to daytime functioning, and no intercurrent hypertension or other cardiovascular disease. These measures can be implemented before testing for sleep apnoea.

Consider the following:

- Weight reduction—as many patients with obstructive sleep apnoea are overweight, weight reduction is generally recommended. Gastric banding or gastric–small intestine bypass operations are generally associated with better weight loss in the long term (currently only available in private hospitals in Fiji). However, if the weight loss is not maintained, the sleep apnoea will return, and the benefit of bariatric surgery will be lost.
- Avoidance of alcohol and drugs that affect sleep.
- Increased time in bed—sleep deprivation together with mild sleep apnoea increases the risk of daytime dysfunction.
- Positional therapy—if the patient has predominantly supine obstructive sleep apnoea, studies have shown that sleeping on their side can improve nocturnal sleep and lessen daytime dysfunction. Preventive measures may include tennis balls attached to the middle of the back of a pyjama shirt, foam blocks to prevent the patient sleeping in the supine position, or alarms that sound if the patient adopts the supine position. Standard pillows behind the back in bed are generally insufficient. Raising the head of the bed (by 5 to 8 cm) so that the bed is angled up may help by reducing ‘rostral fluid shift’, a phenomenon in which

extravascular fluid shifts from lower limbs and abdomen to the thorax, head and neck while the patient is supine overnight.
- Reduced nasal resistance—with smoking cessation and using intranasal corticosteroid spray.

Many complementary treatments (eg nasal strips, nasal dilators, snore-stop drops, snore-easy pillows and other devices) have been marketed to improve or 'cure' snoring and mild sleep apnoea, but they have little supporting scientific data. Nasal expiratory valves have proven benefit, but in general are poorly tolerated.

Continuous positive airway pressure

The most effective therapy for obstructive sleep apnoea is continuous positive airway pressure (CPAP). Consider CPAP for:

- any patient with severe obstructive sleep apnoea (apnoea–hypopnoea index greater than 30)
- patients with mild to moderate obstructive sleep apnoea who have any symptoms of daytime dysfunction
- patients with mild to moderate obstructive sleep apnoea with hypertension or other cardiovascular disease.

CPAP works by splinting the upper airway open. The pressure required to prevent the obstructive apnoea and hypopnoea can be individually titrated, or, automatic titrating machines can be used which monitor airflow during inspiration and adjust the pressure applied.

The table below summarises some common problems that occur with CPAP.

Table 30: Common problems with long-term CPAP

Problem	Possible solution
nasal symptoms due to relatively dry air of CPAP machine	Use intranasal corticosteroid spray, pressure reduction and in-built or add-on humidifiers to CPAP machine.
mouth leak	Use chin straps [Note 1].
dry mouth	Usually indicates significant mouth leak; trial chin straps. Humidification of the CPAP machine will not relieve the dry mouth while a mouth leak exists. Consider reduction in CPAP pressure.
skin ulceration over the nasal bridge	Suggests the mask is not fitted properly or is applied too tightly. Refit the mask or arrange a trial of a new mask.
ear discomfort	Use intranasal corticosteroid spray.

cont...

adherence	Ensure: • mask is comfortable • humidification is operational • there is no anatomic obstruction (eg chronic nasal injury, secondary obstruction) requiring surgical intervention. Exclude coexisting lifestyle, medical and psychological factors that prevent sleep. Provide encouragement. Consider: • using CPAP while distracted (eg while watching TV) • using CPAP on alternate nights • a short nocturnal course of anxiolytic drugs at initiation (eg for 1 week).

CPAP = continuous positive airway pressure

Note 1: Chin straps are especially important when starting CPAP. In the long term, chin straps are important for those patients who have undergone uvulopalatal pharyngoplasty because the normal sealing of the soft palate against the tongue is not possible, increasing the problem of mouth leak.

Obstructive sleep apnoea with other coexisting respiratory disorders

Obstructive sleep apnoea may be associated with chronic obstructive pulmonary disease (COPD), asthma or other significant respiratory disorders.

Patients with severe COPD can develop nocturnal hypoventilation due to a combination of their airway disorder and episodes of apnoea and hypopnoea. It is important to clarify the presence of COPD or other respiratory disorder. To achieve the best outcome for the sleep disorder, it is imperative to optimise daytime respiratory function through weight loss, smoking cessation and appropriate treatment of any underlying respiratory disorder.

Central sleep apnoea

Central sleep apnoea occurs when there is either brief periodic or prolonged loss of respiratory effort during sleep.

Brief periodic loss of respiratory drive is common in:

- heart failure (known as Cheyne-Stokes respiration)
- regular opioid users
- premature infants

- healthy people sleeping at high altitude (higher than 2500 metres).

Central sleep apnoea in these patients is generally associated with a low partial pressure of carbon dioxide (PaCO2).

Prolonged loss of respiratory drive usually indicates a neuromuscular disorder (eg motor neurone disease) or chest wall disorder (eg kyphoscoliosis), or severe obesity (see below). Central sleep apnoea in these disorders is usually associated with elevated PaCO2 or a PaCO2 rise of more than 5 mmHg from the beginning to the end of sleep.

Diagnosis and management of central sleep apnoea is complex and requires specialist care.

Obesity hypoventilation syndrome

Obesity hypoventilation syndrome refers to sleep-related nonobstructive hypoventilation with hypercapnia (partial pressure of carbon dioxide [PaCO2] higher than 45 mmHg) in higher classes of obesity (body mass index [BMI] more than 35 kg/m2) for which no other cause of hypoventilation can be identified (such as kyphoscoliosis, drugs or neuromuscular disease).

For management of obesity see *Fiji Cardiovascular Therapeutic Guidelines:* 'Overweight and obesity' (page 17).

A typical patient with obesity hypoventilation syndrome has the following characteristics:

- marked obesity
- cyanosis or plethora
- right heart failure or biventricular heart failure
- excessive daytime sleepiness.

Many patients exhibit features of both obesity hypoventilation syndrome and obstructive sleep apnoea, with a combination of obstructive apnoeas and progressive hypoxaemia/hypercapnia through the night.

Diagnosis of obesity hypoventilation syndrome requires arterial blood gases taken in the evening and morning. Serum bicarbonate concentration can be used as a marker of prevailing carbon dioxide levels, except in patients with disorders affecting bicarbonate concentration (eg due to kidney failure).

Treatment for obesity hypoventilation syndrome involves weight loss, together with either continuous positive airway pressure (CPAP) or bi-level ventilation with high expiratory pressures. Rarely, in patients with persistent hypoxaemia despite positive airway pressure to assist ventilation, supplemental oxygen may be needed at low-flow rates. Use care to prevent oxygen-induced hypercapnia. Although respiratory stimulants have been used in clinical trials to increase the sensitivity of the ventilatory chemoreceptors, long-term studies to support this therapy are not yet available.

Sleep-disordered breathing in children

Overview

As in adults, sleep-disordered breathing in children may be obstructive sleep apnoea or central sleep apnoea.

Obstructive sleep apnoea occurs in 1 to 4% of children. In most children it is due to adenotonsillar hypertrophy. Other possible causes are listed below.

Central sleep apnoea in children is uncommon.

Table 31: Causes of obstructive sleep apnoea in children

Cause	Comment or examples
adenotonsillar hypertrophy	most common cause
anatomical abnormalities	• macroglossia of Down syndrome [Note 1] • craniofacial abnormalities, eg Robin sequence, Crouzon syndrome
functional abnormalities	• hypotonia of neuromuscular disorders leading to collapse of upper airway during inspiration [Note 1] • hypertonia, commonly seen in cerebral palsy, causing functional narrowing
Obesity	• may be associated with simple obstructive sleep apnoea, with or without tonsillar enlargement • severely obese children may need to be treated as for adults with obesity hypoventilation syndrome

Note 1: In Down syndrome, there may be both anatomical narrowing (due to macroglossia) and functional obstruction (due to hypotonia). It is estimated that 40% of children with Down syndrome have obstructive sleep apnoea.

Diagnosis and clinical assessment

Obstructive sleep apnoea reaches a peak incidence between the ages of 2 to 7 years; it is rare in the first 6 months of life.

The diagnosis is suggested in a child with habitual snoring and observed apnoea during sleep. The box below lists other common nocturnal and daytime features of childhood obstructive sleep apnoea. In contrast to adults, children frequently present with hyperactivity, behavioural problems and poor school performance. Tiredness may or may not be present.

Examination usually reveals tonsillar enlargement and mouth breathing. Children with obstructive sleep apnoea may also present with poor weight gain, which is thought to be related to increased work of breathing during sleep.

A careful history of symptoms of sleep-disordered breathing is warranted in those with predisposing genetic, anatomical or developmental conditions (see table below).

Acute life-threatening events (ALTEs) occur predominantly in infants younger than 6 months. The typical history is of the infant being found pale, limp and not breathing, often during sleep. Vigorous stimulation or active resuscitation has usually been required, and parental anxiety is (not surprisingly) often extremely high. Although acute life-threatening events are thought to have multiple causes, they may be the presenting symptom of sleep-disordered breathing and can occur with obstructive or central sleep apnoea. **Refer all infants who have a significant acute life-threatening event requiring resuscitation for an urgent paediatric assessment to determine if anaetiology can be found.** Some infants may require a sleep study.

Box 10: Clinical features of obstructive sleep apnoea in children

Snoring and/or witnessed apnoeas plus any of the following clinical features may be indicative of obstructive sleep apnoea in a child.

Nocturnal symptoms or signs:

- gasps
- increased work of breathing
- restlessness
- sweating
- night waking
- enuresis
- mouth breathing
- neck extension

Daytime symptoms:

- hyperactivity
- poor attention
- behavioural problems
- poor school performance
- morning hypersomnolence
- tiredness

Investigation and treatment

Most children with a history suggesting obstructive sleep apnoea (see box above) and marked tonsillar hypertrophy are referred directly for adenotonsillectomy without further investigation.

If there is doubt about the diagnosis of obstructive sleep apnoea or the recommended treatment, a further investigation may be necessary. Home overnight oximetry showing multiple clusters of desaturation is a specific but not sensitive test for obstructive sleep apnoea.

Adenotonsillectomy is curative in up to 90% of children with obstructive sleep apnoea in the absence of underlying medical problems. Other treatment strategies, which may be required in the remaining group, include noninvasive ventilation (usually continuous positive airway pressure [CPAP]) or craniofacial surgery. Refer all children with persisting obstructive sleep apnoea following adenotonsillectomy for specialist paediatric assessment.

Central sleep apnoea can occur in children but is far less common than obstructive sleep apnoea. It can only be diagnosed by polysomnography.

Treatment of children with disordered central control of breathing is usually by ventilatory support. There is no place for CPAP in central sleep apnoea. Respiratory stimulants such as caffeine are commonly used in apnoea of prematurity.

Oxygen therapy

Acute oxygen therapy

Principles of oxygen use

Oxygen is a therapeutic agent that may be lifesaving. However, it is not without adverse effects; these include the risk of hypercapnia and adverse consequences related to hyperoxaemia.

Prescribe oxygen on a medication chart or an oxygen prescription form, specifying an appropriate delivery method and flow rates to ensure safe delivery of therapy. The desired outcome is the patient-specific target oxygen saturation range, which must be stated on the prescription. Most oxygenation systems, unlike ventilation systems, do not deliver a precise dose of oxygen at a particular flow rate, with the relative exception of the Venturi mask system and high-flow nasal cannula therapy

Monitor the patient to check the target oxygen saturation is achieved, and to detect adverse effects such as hypercapnia or hyperoxaemia; if needed, adjust the oxygen prescription appropriately.

Prioritise management of the underlying cause of hypoxaemia.

Investigate to determine the cause of hypoxaemia and treat the cause. Recognise that when oxygen is prescribed it is merely treating a result of underlying pathology (ie hypoxaemia) and any need for increased oxygen should prompt a vigorous search for new or worsening pathology.

Potential harms of oxygen use

Possible harms of oxygen use include the risk of potentially fatal hypercapnia and adverse consequences related to hyperoxaemia.

Supplemental oxygen therapy may lead to potentially fatal hypercapnia.

Apart from caution in those at risk of carbon dioxide retention and in premature neonates, avoidance of hypoxaemia and ischaemia has traditionally been emphasised, with little attention focused on the consequences of unrestricted acute supplemental oxygen. Until recently, it was common practice to use oxygen in the clinic, ambulance or emergency department for any patient who appeared breathless or even just acutely unwell.

While there are many beneficial physiological and pharmacological effects of hyperoxia, there is also potential for oxygen toxicity. Reviews have drawn attention to the lack of evidence for improved clinical outcomes with routine use of oxygen in patients who are not actually hypoxaemic. Several clinical studies have provided evidence that supplemental oxygen causes adverse effects in some settings; these include acute myocardial infarction without hypoxaemia or heart failure, acute stroke, and resuscitation of the newborn. A paradigm shift in practice has occurred in response.

Indications

High levels of oxygen are currently recommended for some critical illnesses, including cardiac arrest, shock, major trauma, sepsis, anaphylaxis, status epilepticus, near-drowning, scuba diving accidents, or carbon monoxide poisoningPatients with these illnesses should be given high-flow oxygen through a reservoir mask at 15 L per minute. Once the patient is stable, oxygen should be administered to maintain a target oxygen saturation of 92 to 96%.

Guidelines vary slightly, but there is generally no indication for oxygen therapy in other patients with a pre-treatment oxygen saturation by pulse oximetry of greater than 92 to 96%. In particular, in the absence of hypoxaemia, supplemental oxygen is no longer recommended for acute coronary syndromes, stroke, or obstetric emergencies. Neonatal resuscitation guidelines target much lower oxygen saturations for much longer than previously recommended before advising a switch from room air to supplemental oxygen. One caveat is that pulse oximetry readings may be misleading in some situations (see below).

Other acutely unwell patients, many of whom will have an underlying respiratory cause, need moderate levels of supplemental oxygen if the patient is hypoxaemic on pulse oximetry. Oxygen may initially be administered by nasal cannulae at 2 to 4 L per minute or a simple face mask at 5 to 10 L per minute. However, if the oxygen saturation is less than 85% and the patient is not at risk of hypercapnic respiratory failure (see below), oxygen should be started and titrated to effect as the situation stabilises.

Image 8: An example of a nasal cannula

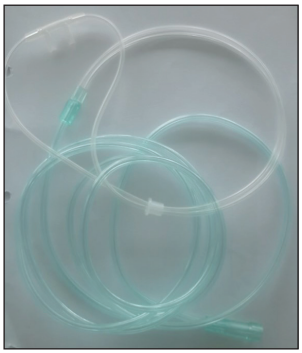


Image 9: An example of a reservoir mask

Patient groups at risk of hypercapnia

Identify patients at risk for hypercapnic respiratory failure if oxygen therapy is being considered; these patients may hypoventilate and develop a rise in carbon dioxide levels. To mitigate this potentially fatal risk, use lower target oxygen saturation with appropriate monitoring. Recognise that adequate oxygenation is not the same as adequate ventilation, and that pulse oximetry does not detect hypercapnia.

The following conditions or situations may put patients at risk of hypercapnia:

- chronic obstructive pulmonary disease (COPD) or bronchiectasis
- history of heavy smoking
- severe obstructive sleep apnoea
- morbid obesity
- severe kyphoscoliosis or ankylosing spondylitis

- neuromuscular disorders with respiratory muscle weakness, especially if using home ventilation
- use of respiratory depressant drugs such as opioids and benzodiazepines
- acute asthma in some patients.

Note that the above list is not exhaustive.

Except for patients with recurrent hypercapnic respiratory failure, it is not possible to predict if an individual patient with COPD will develop hypercapnia during an acute exacerbation; therefore, consider all patients with moderate to severe COPD at risk of hypercapnia. Once identified, patients who have developed hypercapnic respiratory failure with oxygen should be given some form of documentation such as a medical alert bracelet.

Target oxygen saturation

The target oxygen saturation is generally 92 to 96% unless the patient has an underlying condition that results in chronically lower values or risk of hypercapnic respiratory failure. For the latter group, oxygen should be given to achieve a target oxygen saturation of 88 to 92%; however, in critical illness, the standard target oxygen saturations (92 to 96%) may initially be used pending the results of early arterial blood gas analysis.

Monitoring

With acute oxygen therapy, monitor pulse oximetry continuously. Alter the flow administered to keep the oxygen saturation within the target range.

Be aware of the limitations of pulse oximetry. It measures only haemoglobin saturation, not oxygenation or ventilation. Readings may also be misleading, for example they will be misleadingly low with hypoperfusion and methaemoglobinaemia but conversely falsely reassuring in carbon monoxide poisoning, cyanide poisoning and severe anaemia. In particular, pulse oximetry does not measure carbon dioxide levels; arterial blood gases must be performed in patients thought to be at risk of hypercapnia.

Arterial blood gas analysis is needed to quantify hypercapnia; it is not detected by pulse oximetry.

Monitoring of oxygen therapy is an ongoing process. Repeat arterial blood gases are needed if the patient's condition changes (for example drowsiness suggesting hypoventilation) or about 30 to 60 minutes after oxygen flow rates have been increased.

In addition, serial blood gases may be indicated at routine intervals to track the patient's progress in those who are critically unwell. If blood gas values for oxygen

and carbon dioxide cannot be corrected satisfactorily with oxygen therapy, ventilatory support may be indicated.

Domiciliary oxygen therapy

Domiciliary oxygen therapy is prescribed for patients with chronic hypoxaemia, with the most common cause being chronic obstructive pulmonary disease (COPD) in adults, and chronic neonatal lung disease in children.

Domiciliary oxygen is not prescribed to treat the symptom of breathlessness without hypoxaemia. The purpose of domiciliary oxygen therapy is to prolong life.

Oxygen therapy can be administered either intermittently or continuously depending on the condition being treated and the patient's ability to meet the need.

Domiciliary oxygen therapy is indicated for children with severe chronic hypoxaemia (unable to maintain oxygen saturation measured by pulse oximetry [SpO2] greater than 94% in room air). The largest group of children with severe chronic hypoxaemia are infants with chronic lung disease of prematurity, which is a sequela to severe hyaline membrane disease (caused by deficiency of pulmonary surfactant).

Long-term continuous oxygen therapy has been shown to prolong survival in adult patients with:

- stable chronic obstructive pulmonary disease (COPD) who, when breathing air at rest and awake, have a consistent partial pressure of oxygen (PaO2) of 55 mmHg or less
- stable COPD with evidence of complications of hypoxaemia, such as polycythaemia, pulmonary hypertension or right-sided heart failure, and a PaO2 of 59 mmHg or less.

The goal of oxygen therapy is to maintain a resting oxygen saturation measured by pulse oximetry (SpO2) greater than 90% during periods of wakefulness. This is usually achieved at an oxygen flow of 2 L per minute. Patients needing continuous domiciliary oxygen should use oxygen supplementation for as close as possible to 24 hours per day, ideally a minimum of 18 hours per day; this includes hours asleep.

There is equivocal evidence for benefit of intermittent oxygen therapy. It may be indicated for patients who have obstructive or fibrotic lung diseases who do not meet the criteria for long-term continuous oxygen therapy and whose ability to exercise is limited by hypoxaemia.

Intermittent oxygen therapy may be prescribed also for some patients with severe asthma and for palliative relief of dyspnoea due to hypoxaemia in some irreversible chronic lung conditions such as interstitial lung disease and neoplastic lung disease.

In some patients only short-term therapy is required, hence a reassessment of therapy is advisable in 4 to 8 weeks.

Long-term and intermittent domiciliary oxygen are not indicated for patients:

- whose main complaint is dyspnoea but who maintain PaO2 higher than 60 mmHg
- who continue to smoke (because of the risk of causing fire)
- who have not received adequate therapy for their underlying disease.

Oxygen is usually delivered by oxygen concentrators or cylinders. Choice of delivery system depends on cost and the number of hours per day that oxygen is needed.

For domiciliary oxygen therapy, low-flow devices are usually prescribed. The first-line choice is the standard nasal cannula; however, consider the patient's clinical condition, PaO2, PaCO2 and comfort when choosing the device.

Oxygen concentrators are a cost-effective means of delivering oxygen to patients needing continuous therapy.

Adverse effects of oxygen delivery

Nasal symptoms from oxygen delivery, such as nasal stuffiness, dryness or bleeding, are common and may be alleviated by the use of saline nasal sprays (sodium chloride solution) or water-based lubricant, or by placing the cannulae in the mouth, which may be practical at night. Nasal cannulae may cause pressure area marks behind the ears or on the cheeks, which can be avoided and alleviated by the use of soft foam pads, cotton wool or gauze.

The equipment supplier may be able to advice on the availability of extra devices or attachments that would help to solve some of these problems.

For details regarding the indication and availability of oxygen therapy, please contact the physician at the nearest divisional hospital.

Noninvasive ventilation

Introduction

Noninvasive ventilation (NIV), also known as noninvasive positive pressure ventilation (NIPPV), is ventilatory support given by a face mask rather than by endotracheal intubation. It is an effective means of treating patients with acute respiratory failure, particularly in exacerbations of chronic obstructive pulmonary disease (COPD), acute pulmonary oedema and immunocompromised patients.

Patients must be conscious enough to protect their airway and have intact airway reflexes.

Noninvasive ventilation may consist of continuous positive airway pressure (CPAP) or bi-level therapy (BiPAP), where a higher pressure is given during inspiration and lower during expiration.

Noninvasive ventilation is supportive therapy to be used only in addition to first-line therapy directed at the underlying disease.

Sufficient adequately trained staff are needed to give and monitor noninvasive ventilation, usually in a critical care environment or a high-dependency unit. Arterial blood gases should be monitored.

Indications for acute noninvasive ventilation

Acute exacerbations of COPD

Patients particularly suitable for noninvasive ventilation are those with exacerbations of chronic obstructive pulmonary disease (COPD) with carbon dioxide retention, respiratory acidosis with a pH in the range 7.25 to 7.35, and tachypnoea. In this group, it reduces the need for intensive care admission as well as length of hospital stay and mortality. It is also useful for relieving their dyspnoea.

Noninvasive ventilation should also be trialled in patients with more severe acidosis; however, if it fails they may need tracheal intubation and invasive mechanical ventilation. Usually in these more severely acidotic patients, the trial is best done in a critical care environment, except if noninvasive ventilation is the agreed ceiling of care. Before instituting any form of ventilatory support, a decision should be made as to what the ceiling of care is and whether intensive care unit (ICU) admission is contemplated.

Acute cardiogenic pulmonary oedema

Continuous positive airway pressure (CPAP) (at a pressure of 5 to 10 cm H2O) is useful in acute cardiogenic pulmonary oedema. Noninvasive ventilation has been shown to produce more rapid improvement in respiratory distress and metabolic disturbance than standard therapy, but has no effect on short-term mortality.

Hypoxaemic respiratory failure

Noninvasive ventilation may be beneficial for patients with hypoxaemic respiratory failure caused by conditions other than COPD or acute pulmonary oedema, particularly for immunocompromised patients (eg neutropenic patients with pulmonary infiltrates) or patients with leptospirosis and acute pulmonary haemorrhage. However, patients must be able to protect their airway and their respiratory rate must settle noticeably (to less than 30 breaths per minute) when NIV applied. If not, invasive ventilation is likely more appropriate.

Weaning from invasive ventilation

Weaning with noninvasive ventilation should be beneficial for intubated and mechanically ventilated ICU patients in whom the risk of post-extubation respiratory failure is high. These patients often have pre-existing respiratory or cardiac disease or have been in ICU for long periods of time (more than 10 days), often recuperating following a sudden medical or surgical event.

Acute asthma

Noninvasive ventilation may be used in acute asthma, but caution is needed and close observation in a high-dependency or intensive care unit is recommended.

Non-invasive ventilation must be compatible with continuous administration of salbutamol nebules.

Contraindications to noninvasive ventilation

Use of noninvasive ventilation is contraindicated in the following situations:

- impaired consciousness with inability to protect the airway
- immediate need for tracheal intubation
- cardiorespiratory arrest
- haemodynamic instability
- fixed upper airway obstruction
- copious secretions or vomiting
- pneumothorax

- facial injuries
- recent upper gastrointestinal surgery
- uncooperative patient or patient intolerant of the mask
- insufficient trained staff to give and monitor noninvasive ventilation.

Using noninvasive ventilation

Before using noninvasive ventilation

Measure arterial blood gases, where possible, *before* commencing noninvasive ventilation.

Obtain informed consent and discuss whether noninvasive ventilation is the ceiling of care or whether intubation will be undertaken if noninvasive ventilation fails.

Set up and check the appropriate ventilator mask and circuit, which must include inspiratory and expiratory limbs or a carbon dioxide exhalation port.

Equipment settings and monitoring

Equipment settings and monitoring for noninvasive ventilation are listed in the box below. Either a full-face or nasal mask can be used.

Once the machine is attached and switched on, hold the mask to the patient's face to familiarise them with it. After a few minutes the headgear can be secured. Do not overtighten the headgear because severe ulceration of the bridge of the nose can occur very rapidly.

Show the patient how to remove the mask and how to summon help if needed.

Box 11: Equipment settings and monitoring for noninvasive ventilation

Suggested settings for noninvasive ventilation:

- Initial settings for bi-level noninvasive ventilation in COPD are inspiratory positive airway pressure (IPAP) 4 to 6 cm H_2O and expiratory positive airway pressure (EPAP) 4 to 6 cm H_2O.
- If a spontaneous/time mode ventilator is used, set this with a backup rate of 15 breaths per minute and inspiratory to expiratory (I:E) ratio of 1:3.
- Set triggers at maximum sensitivity.
- Once patient is comfortable on initial settings, IPAP is usually increased to 10 to 12 cm H_2O and EPAP may also be increased if needed.
- Initial settings for CPAP in acute pulmonary oedema are 5 to 15 cm H_2O (approximately 10% of body weight in kg).

Suggested monitoring for noninvasive ventilation:

- Monitor pulse oximetry continuously.
- Reassess the patient within a few minutes and adjust ventilator settings and supplemental oxygen to optimal levels
- Repeat clinical assessment and arterial blood gases in 1 hour and thereafter as clinically indicated.

COPD = chronic obstructive pulmonary disease; CPAP = continuous positive airway pressure

Ventilator adjustment and supplemental oxygen

Expiratory positive airway pressure (EPAP) improves aeration of the lungs and, in chronic obstructive pulmonary disease (COPD), overcomes intrinsic positive end expiratory pressure (PEEPi). The main indication for increasing EPAP is persistent hypoxaemia despite a satisfactory fall in partial pressure of carbon dioxide (PaCO2). Inspiratory positive airway pressure (IPAP) provides pressure support for inspiration, which increases alveolar ventilation and assists in reducing carbon dioxide.

For patients receiving noninvasive ventilation, adequacy of ventilation is assessed clinically by assessing chest expansion, and biochemically by repeat arterial blood gases and/or the device's measurements. Once the patient is comfortable on non-invasive ventilation, there is usually a difference of at least 6 cm H2O between IPAP and EPAP. Supplemental oxygen should be given after optimising ventilator settings to maintain the oxygen saturation measured by pulse oximetry (SpO2) between 88 to 92% for patients with COPD and 90 to 95% for patients with acute pulmonary oedema.

In some patients with mainly hypoxaemic respiratory failure, high-flow oxygen therapy can be considered because it provides heated and humidified oxygen blends (21 to 100%) with 2 to 3 cm H2O continuous positive airway pressure (CPAP) via loose-fitting nasal prongs.

Management of problems

Treatment failure

Treatment failure with noninvasive ventilation may be indicated by clinical deterioration, increasing distress or deteriorating arterial blood gas results.

If treatment failure occurs:

- ensure the medical treatment has been optimised (bronchodilators, corticosteroids, antibiotics) and ensure it has actually been given
- consider chest X-ray to exclude pneumothorax or aspiration pneumonia
- consider physiotherapy for sputum retention.

If there is persistent elevation of the PaCO2 and continued acidosis:

- check inspired oxygen concentration and pulse oximetry and reduce oxygen if necessary. Target oxygen saturation measured by pulse oximetry (SpO2) is 88 to 92% for patients with chronic obstructive pulmonary disease (COPD)
- check the circuit for leaks, including mask leaks, and check that the circuit includes a carbon dioxide expiration port
- consider increasing expiratory positive airway pressure (EPAP) to reduce the possibility of rebreathing
- check the patient's synchronisation with the ventilator and adjust the backup rate
- check the ventilator trigger is set for maximum sensitivity
- observe chest expansion and, if inadequate, consider increasing inspiratory positive airway pressure (IPAP). If this is not tolerated or chest expansion is adequate, consider increasing respiratory rate or inspiratory to expiratory (I:E) ratio to increase expiratory time.

If there is persisting hypoxaemia despite improvement in PaCO2, consider increasing EPAP (maintain the difference between IPAP and EPAP) or increasing supplemental oxygen, and review the need for intubation and mechanical ventilation. If the patient remains tachypnoiec and distressed on non-invasive ventilation then, unless a ceiling to treatment has been agreed, progressing to invasive ventilation would be appropriate.

Miscellaneous problems

Some miscellaneous problems associated with noninvasive ventilation are:

- nasal bridge ulceration, which should not occur with appropriate mask fitting. A prophylactic hydrocolloid dressing may be used
- rhinorrhoea, which may be reduced by the use of ipratropium nasal spray and by incorporating a heated humidifier in the circuit
- retention of secretions, which may be overcome by humidification, bronchodilators and regular physiotherapy

- gastric distension, which may cause discomfort. Consider lowering both IPAP and EPAP.

Stopping noninvasive ventilation

In the acute setting, noninvasive ventilation usually produces rapid improvement. In exacerbations of chronic obstructive pulmonary disease (COPD) it is usually applied throughout the first 24 hours (with breaks for meals) and then overnight for 1 to 2 nights. For patients with cardiogenic pulmonary oedema, continuous positive airway pressure (CPAP) is usually only required for approximately 6 to 12 hours.

Patients can usually be weaned from noninvasive ventilation once dyspnoea has resolved and arterial blood gases have normalised. Following acute noninvasive ventilation, consider assessing ventilation during sleep (eg by pulse oximetry) to guide whether overnight noninvasive ventilation should be continued for a few extra nights to allow restoration of quality sleep. Most patients admitted to hospital with acute-on-chronic respiratory failure have arrived severely sleep deprived.

Fitness for surgery

Patients who have respiratory impairment due to respiratory disease need proper assessment before surgery. They may require specific interventions or precautions before and after the operation.

Nature of the risks

In general, deficiencies of the respiratory system can be overcome during surgery by using assisted ventilation, high concentrations of supplemental oxygen and effective intra-airway suction to remove secretions. However, the postoperative period is a time of major morbidity and mortality for patients with respiratory disease. This is due to increased demands on, and reduced capacity of, the respiratory system.

Increased demands on the respiratory system, such as fever, sepsis and tissue repair, cause the basal metabolic rate to increase two- to three-fold postoperatively. This increases oxygen consumption and carbon dioxide production, and hence the requirement for increased ventilation.

Reduced ventilatory capacity of the respiratory system postoperatively can be due to pain from abdominal and thoracic wounds, the need to lie supine and the use of sedatives and analgesics. It can also be due to atelectasis, sputum retention, pneumonia and fluid overload adversely affecting both gas-exchange and ventilatory capacity.

Physiotherapy review is important for prevention of post-operative complications in all patients, but particularly those at risk of complications.

Risk groups

The risk of perioperative complications depends on the type of procedure and the nature of the respiratory disease.

Procedures that put patients with respiratory disease at most risk are:

- coronary artery bypass grafts and procedures involving the thorax and upper abdomen
- procedures with long operating duration
- procedures requiring high levels of postoperative analgesia.
- head and neck surgery

People with respiratory disease at most risk of postoperative complications are those:

- who smoke
- with poorly controlled asthma and/or a history of severe flare-ups (exacerbations)
- taking long-term systemic or high-dose inhaled corticosteroids; a brief course of supplementary corticosteroids may be needed perioperatively
- with limited mechanical ventilatory reserve, eg due to
  - severe airway obstruction
  - interstitial lung disease
  - obesity
  - disease associated with weak respiratory muscles
- with mucus hypersecretion, eg due to
  - chronic bronchitis
  - bronchiectasis
  - cystic fibrosis
- with a chest wall disorder causing a rigid chest, eg kyphoscoliosis
- with reduced ability to protect the upper airway or to clear secretions from the lungs, eg due to neuromuscular disorders affecting the bulbar muscles and cough mechanism
- with uncontrolled gastro-oesophageal reflux
- prone to pulmonary oedema because of coexisting cardiac disease
- with significant sleep apnoea
- prone to respiratory centre depression, eg due to chronic carbon dioxide retention
- who have difficulty cooperating with instructions, eg with physical or mental disability.
- advanced age (over 70 years)

In addition, some patients are deemed by anaesthetists to have an airway that is difficult to intubate. These patients often have significant craniofacial abnormalities such as a small mouth opening or significant retrognathia. They are more likely to develop postoperative respiratory complications and are more prone to obstructive sleep apnoea. The STOPBang questionnaire (available from the British Snoring and Sleep Apnoea Association website www.britishsnoring.co.uk/stop_bang_questionnaire.php) may help to identify obstructive sleep apnoea; these patients may benefit from continuous positive airway pressure (CPAP) or noninvasive ventilation postoperatively. In addition, patients with OSA have a higher risk of difficult airway.

Acute viral and bacterial infections can temporarily affect mucociliary function and increase mucus production and increase bronchial reactivity. This can increase the risk of intraoperative and postoperative complications. In otherwise healthy people,

this risk is relatively low but present and is influenced by many factors including type of anaesthetic and procedure being performed.

Children who have had a viral respiratory tract infection in the preceding 4 weeks are at increased risk of intraoperative respiratory events (coughing, laryngospasm, oxygen desaturation), but there is no evidence of increased postoperative morbidity or mortality. It is unnecessary and impractical to cancel surgery for children with a history of a recent respiratory tract infection. However, elective surgery should be delayed for 2 to 4 weeks if a child is acutely viraemic (eg has fever, malaise, lack of appetite, rhinorrhoea).

For elective surgery the decision to proceed in the setting of respiratory infection requires anaesthetist review, risk assessment, and informed patient consent.

Assessment

The assessment of fitness for an anaesthetic in individual patients with an acute illness rests with the anaesthetist.

Clinical assessment

Clinical assessment of a patient before surgery should include relevant assessment of:

- smoking—use a multifaceted approach to quitting and, if practical, postpone surgery until the patient has not smoked for 6 weeks (see Australian and New Zealand College of Anaesthatists (ANZCA) Guidelines on Smoking as Related to the Perioperative Period available from http://www.anzca.edu.au/documents/ps12-2013-guidelines-on-smoking-as-related-to-the.pdf)
- evidence of pulmonary hypertension or right-sided heart failure
- chronic bronchitis and bronchiectasis—take measures to improve mucus clearance
- unstable asthma—take measures to control
- respiratory impairment—usually identified because of breathlessness and reduced exercise capacity. This may be due to asthma, chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, pulmonary vascular disease or respiratory muscle weakness.
- respiratory infection

Respiratory function tests

All patients with clinical evidence of respiratory impairment, should have their respiratory function tests (such as spirometry or cardiopulmonary exercise testing) performed if available. If a patient is unable to perform respiratory function testing

due to physical or mental disability, in addition to careful postoperative monitoring, it may be necessary to use surrogates such as thorough clinical examination, chest X-ray, pulse oximetry and blood tests to assess for hypercapnia (or serum bicarbonate concentration) and polycythaemia.

If oxygen therapy is provided, it is as important to monitor for and prevent hyperoxaemia as it is to treat hypoxaemia.

No further respiratory assessment is needed for patients with both a forced expiratory volume in 1 second (FEV_1) and a diffusing capacity for carbon monoxide (DLCO) of greater than 60% of predicted because they have a low risk of postoperative complications (even following major operations such as pneumonectomy).

Lobectomy is associated with a low risk of postoperative complications if FEV_1 is greater than 40% of predicted and DLCO is greater than 60% of predicted. This probably also applies to other relatively high-risk operations such as chest or upper abdominal surgery.

The lower limit of respiratory function needed for medium- or low-impact surgery (ie not interfering with respiratory muscles or cough) is not known. However, an individual with any of the following has a very limited respiratory reserve and needs expert preoperative assessment:

- FEV_1 less than 60% of predicted
- DLCO less than 60% of predicted
- partial pressure of carbon dioxide (PaCO2) higher than 45 mmHg
- moderate or severe pulmonary hypertension
- oxygen saturation measured by pulse oximetry (SpO2) 90% or less when breathing room air
- receiving long-term domiciliary oxygen.

Postoperative management

The key requirements in the early postoperative period for patients with respiratory impairment are:

- good pain control (epidural analgesia may be indicated). Supplemental oxygen, humidified for 'mouth breathers.' Ensure supplemental oxygen is titrated to oxygen saturation measured by pulse oximetry (SpO2), eg 92 to 96% in otherwise healthy patients, 88 to 92% in patients prone to hypercapnia such as patients with chronic obstructive pulmonary disease (COPD)
- nebulised bronchodilators. Take care to avoid excessive dosing, eg more frequently than 4 hourly
- prevention of atelectasis by effective deep breathing and coughing, and prophylactic continuous positive airway pressure (CPAP) or bi-level support in

some instances, eg after cardiac surgery or in children with neuromuscular disorders or restrictive lung disease such as kyphoscoliosis
- airway clearance techniques, eg bubble positive airway pressure (PEP)
- early ambulation
- increased corticosteroid dosage in appropriate situations, eg patients with asthma.

Necessary prevention or early identification of potential complications involves:

- frequent clinical examination to detect fever, inspiratory crackles, disorientation
- continuous or regular monitoring of SpO2 and partial pressure of carbon dioxide (PaCO2) if indicated. Ensure avoidance of excessive oxygen, thus keeping SpO2 at 92 to 96% in otherwise healthy patients, or at 88 to 92% if patients are prone to hypercapnia, eg patients with COPD
- staff being alert to the dangers of oversedation, vomiting and aspiration, reflux, fluid overload and cardiac arrhythmias
- formal monitoring of pain control and the patient's ability to cough effectively
- monitoring sputum volume and purulence
- monitoring forced expiratory volume in 1 second (FEV_1) and/or peak expiratory flow when appropriate.

Abnormal chest findings, fever or hypoxaemia warrant investigation, including a chest X-ray.

Fitness to fly

Introduction

Air travel is safe for the vast majority of the two billion or so passengers who fly each year. It can pose significant risks to people with respiratory disease, although most tolerate flight well. Approximately 12% of acute in-flight medical episodes are respiratory in nature, and it is likely that respiratory disease and hypoxaemia contribute to other episodes including cardiac or neurological events.

No further investigation is needed for patients whose respiratory disease is stable when assessed, who have not had a previous in-flight problem, and whose resting arterial blood oxygen saturation is 95% or greater. For other patients with respiratory disease, spirometry, arterial blood gas and haemoglobin oxygen saturation levels do not reliably predict in-flight complications, so further assessment is necessary.

Cabin environment

Commercial aircraft usually cruise at altitudes between 10 000 and 13 500 metres (32 800 and 44 300 feet). Pressurisation of the cabin ensures that the pressure corresponds to an altitude much lower than that at which the aircraft is flying. However, for reasons of aircraft weight and fuel economy, the cabin is not fully pressurised to sea level.

The maximum allowable cabin altitude by international regulation is 2438 metres (8000 feet), generating a cabin air pressure of 565 mmHg. At this pressure, the partial pressure of inspired oxygen (PiO2) in humidified air falls to 108 mmHg (normally about 148 mmHg at sea level), which is equivalent to breathing 15.1% oxygen at sea level (normally 20.8%). The low PiO2 causes a reduction in arterial oxygen partial pressure (PaO2). In people with normal lung function, the PaO2 is likely to fall to between 53 and 64 mmHg with haemoglobin oxygen saturation (SaO2) falling to 85 to 92% (normally greater than 95%). Healthy people do not notice this change.

In people with chronic respiratory disease who already have a reduced PaO2 and SaO2 at sea level, the reduced PiO2 may cause significant hypoxaemia and reduced tissue oxygenation.

In addition, the reduced cabin pressure causes expansion of gas by about one-third. This can be important if there is trapped gas in closed body spaces such as the sinuses, middle ear, pleural cavity or pulmonary bullae.

Other features of commercial air travel, which might cause problems, are:

- dryness of the air
- reduced mobility of travellers, causing potential for venous thromboembolism
- close proximity to other people and consequent risk of respiratory tract infections
- disturbance of circadian rhythms on longer flights

Pre-flight assessment

Assessing the need for in-flight supplemental oxygen

Patients needing assessment

Patient groups needing assessment for in-flight supplemental oxygen include those:

- with a history of respiratory problems during air travel, such as breathlessness, chest pain, confusion or syncope
- with severe asthma or severe chronic obstructive pulmonary disease (COPD) (forced expiratory volume in 1 second [FEV_1] less than 30% predicted)
- with severe restrictive lung disease (forced vital capacity [FVC] less than 1 Litre)
- with arterial oxygen saturation measured by pulse oximetry (SpO2) of less than 95%
- within 6 weeks of hospital discharge for acute severe or acute-on-chronic respiratory illness
- with a comorbidity that is worsened by hypoxaemia, such as cerebrovascular disease, coronary artery disease or heart failure
- with a pre-existing requirement for supplemental oxygen or ventilator support, including noninvasive ventilation.

Clinical assessment

Clinical assessment includes cardiorespiratory history and examination, spirometry, pulse oximetry and review of previous air travel experience.

Hypoxic challenge test

The hypoxic challenge test (HCT), also known as the high-altitude simulation test (HAST), simulates the reduced inspired oxygen partial pressure (PiO2) at 2438 metres (8000 feet) by exposing the person to air containing a reduced concentration of oxygen at sea level. The test is performed in a respiratory function laboratory over a 20- to 30-minute period, either with special low-oxygen gas cylinders or with the person breathing air while wearing a 40% Venturi mask with nitrogen as the driving gas.

Arterial oxygenation is assessed by continuous measurement of oxygen saturation by a pulse oximeter (SpO2). If SpO2 falls below 85% or the patient becomes distressed during HCT, in-flight oxygen is indicated.

The HCT has been validated and has the advantage that the effects of any hypoxaemia, such as respiratory distress or angina, can be observed. However, it is too complicated to use as a screening test in large populations.

Walk tests

Walk tests are no longer recommended unless hypoxic challenge testing is unavailable. Patients unable to walk 50 metres on level ground should be considered unfit to fly.

Assessment of other respiratory conditions

Doctors in primary care are often asked whether a child or adult with an acute respiratory tract infection is fit to fly. Upper airway infections can impair the ability to equalise pressures between the middle ear and oropharynx via the eustachian tube. This can result in barotrauma to the tympanic membrane, particularly at the time of descent. If travel is necessary, and the patient has lost their usual ability to equalise the pressure with the Valsalva manoeuvre at sea level, nasal or oral decongestants may be used at a suitable time before ascent and/or descent. Anecdotal evidence suggests that semi-permeable earplugs (eg Ear Planes, Alpine FlyFit) may be helpful.

Flying with an upper respiratory tract infection rarely causes permanent damage to the tympanic membrane. However, patients who experience deafness, vertigo, or bleeding from the external auditory meatus after air travel must be examined by their doctor.

As with any infection, patients with an acute respiratory tract infection should be cautioned about the risk of spreading the infection to others.

Patients with pulmonary tuberculosis must not travel by air until they are noninfectious (usually after at least 2 weeks of effective treatment).

The volume of air in the pleural space expands by about one-third at cabin altitude, so patients who have a pneumothorax should not travel by air unless they have an intercostal drain with a one-way valve (Heimlich valve) in place. Air travel should be delayed for 1 week after radiological resolution of spontaneous pneumothorax or by 2 weeks following pneumothorax after thoracic surgery or trauma.

Reports of problems during air travel in patients with large lung bullae are rare. Pressure changes during commercial flights happen slowly, allowing time for equilibration, so the presence of bullae is not a contraindication to air travel.

Infants and young children who have had chronic neonatal lung disease require specialist assessment before air travel, even if they no longer require supplemental oxygen.

In-flight oxygen therapy

The usual flow rate for in-flight oxygen is 2 L per minute via nasal cannulae. Patients already using supplemental oxygen should have their normal flow rate increased. Those needing oxygen at flow rates of 4 L per minute or more at sea level are not fit to fly.

The requirement for in-flight oxygen must be communicated to the airline well in advance of travel, often by completion of a medical information form, which details the patient's condition and oxygen requirements eg Fiji Airways Medical Form: www.fijiairways.com/media/140051/medical-form.pdf.

Most airlines provide in-flight oxygen on request and may charge for this service. Consider the need for oxygen at the airport (including for stopovers) because most airlines only provide in-flight oxygen. Some airlines allow passengers to carry and use their own small oxygen cylinders on board.

In-flight continuous positive airway pressure

For most people with obstructive sleep apnoea, it is not essential to use continuous positive airway pressure (CPAP) every time they sleep. However, CPAP may be required by some patients with severe obstructive sleep apnoea on long-haul flights.

If CPAP is necessary, the airline should be consulted before making the travel booking. A doctor's letter outlining the diagnosis and necessary equipment is needed. It should state the CPAP machine should travel in the cabin as extra hand luggage. Dry cell battery–powered CPAP can be used during the flight but must be switched off before landing.

During the flight, all patients with obstructive sleep apnoea should avoid factors that worsen their apnoea, such as alcohol or sedatives.

Fitness to scuba dive

Introduction

All professional scuba divers in Fiji are required to undergo regular examinations for medical fitness. Most diving organisations require trainee recreational scuba divers to undergo a medical examination or declare any relevant medical conditions. The standards suggested by the South Pacific Underwater Medicine Society (SPUMS) have been adopted as the Fiji standard. There is evidence that, in practice, these standards are not always applied.

Introductory or 'resort' dives can be performed without a medical examination. Divers are required to fill in a questionnaire and to declare any relevant conditions. If there is any doubt, divers should be referred for full assessment.

Pre-diving medical examinations approved training course in underwater medicine can be obtained from SPUMS <www.spums.org.au>.

Pulmonary barotrauma is the most common cause of severe diving accidents, and most of the decisions regarding fitness to scuba dive involve assessment of the respiratory system. While scuba diving, the diver breathes air at the same pressure as the surrounding water. As the diver ascends, the air in the lungs expands (in accordance with Boyle's law) and, unless it can escape from the air spaces easily, there is the potential for barotrauma (pneumothorax, pneumomediastinum and arterial gas embolism).

Pressure changes on ascent are proportionally greater at shallower depths. Consequently, with respect to altered respiratory structure or function, there is never an indication to pass divers as fit with the proviso that they do not exceed a certain depth.

A reasonable overall level of cardiorespiratory fitness is required for safe scuba diving. Even if candidates meet other more specific criteria, if general physical fitness and cardiorespiratory reserve seem inadequate to cope with potential emergencies, they should be advised against diving.

Analyses of scuba diving fatalities show that medical conditions are rarely implicated, with the exception of sudden cardiac deaths. Most accidents are caused by poor training and unsafe diving practices, not by illness.

Respiratory conditions affecting fitness to scuba dive

Asthma and other obstructive airway disease

Carefully assess people with significant obstructive airway disease because there is a theoretical risk of localised gas trapping due to airway narrowing or the presence of bullae, resulting in an increased risk of barotrauma. Most of these patients are disqualified on spirometric criteria (see Assessment below). However, those who meet the criteria should be further assessed for exercise tolerance and the presence of other smoking-associated diseases that may render them unfit to dive.

The most contentious decisions regarding fitness to dive relate to the presence or absence of asthma. A diagnosis of asthma has been an automatic disqualification in the past, but attitudes have changed and well-controlled asthma is now regarded as compatible with diving (as it always has been in many countries).

People with asthma who are asymptomatic and who have normal lung function, even if on regular treatment, may be fit to dive. However, consider that many people are given asthma medication for uncertain indications and they may not have asthma.

For patients with a history of asthma but no current medication use, the standards state that further investigation is needed, including bronchial provocation testing with exercise, hypertonic saline (sodium chloride solution), histamine, methacholine or mannitol. Bronchial provocation testing is also recommended if the patient has had asthma symptoms within the last 10 years. Those who fail bronchial provocation testing may be retested after asthma therapy has been optimised. Annual retesting is advised.

There is no evidence of a statistically significant risk of cerebral gas embolism or other diving accidents in people with asthma who scuba dive. Rather than harm from barotrauma, the major risk of harm is probably related to the development of a flare-up of asthma triggered by exercise, inhalation of cold dry air or aspiration of sea water, resulting in diminished exercise capacity and a higher risk of drowning. Swimmers, surfers and snorkellers may be at similar risk. People with wheeze precipitated by exercise or cold should be advised not to dive.

The use of a scuba regulator during exercise in people with asthma is associated with significant decreases in forced expiratory volume in 1 second (FEV_1).

The widely held belief that diving regulators can develop faults and produce a stream of respirable-sized particles of salt water, and hence precipitate bronchospasm, is almost certainly untrue.

Pneumothorax

Any history of spontaneous pneumothorax precludes scuba diving because of the almost certain presence of bullae or blebs on the visceral pleura. Lung bullae increase the risk of barotrauma and are a contraindication to diving.

Although there is no evidence of increased risk, the standards also state that previous perforating chest injuries or open chest surgery are automatic disqualifications. Chest surgery (such as coronary artery surgery) where the pleural cavities are not entered is not specifically a disqualification.

Patients with a history of traumatic pneumothorax not involving chest wall perforation are also not specifically excluded from diving by the standards but could be at increased risk because of local lung scarring. Some authorities recommend expiratory chest X-rays or high-resolution computed tomography (HRCT) scans of the lung in such cases, but there is no sound basis to this. If there are residual abnormalities on the plain chest X-ray, the patient should be declared unfit to dive.

Upper respiratory tract problems

The most common upper respiratory tract problems in scuba divers are infection, allergic rhinitis and sinus disease. On descent and ascent, pressure of air in the middle ear and in the sinuses has to equalise with the ambient pressure; failure of pressure to equalise leads to pain (squeeze) and, potentially, to barotrauma of the ear. If a diver has upper respiratory tract inflammation related to infection or allergy, which can interfere with pressure equalisation, they should not dive until their symptoms resolve. It is common practice among divers to take decongestants such as pseudoephedrine in such situations, but this should be discouraged.

Other conditions

The standards state that any chronic lung disease or fibrotic lesion of the lung that may cause altered compliance in lung tissue, including cystic fibrosis, should disqualify the person from diving. This lacks any supportive evidence and is based on theoretical considerations.

There is a single case report of decompression illness related to asymptomatic sarcoidosis.

Patients with active pulmonary tuberculosis should not dive.

Assessment

The patient history should include details of exercise tolerance, smoking history, current symptoms, childhood symptoms that may suggest asthma, upper airway problems and any episodes that could indicate previous pneumothorax.

Spirometry, including a flow–volume loop, must be performed on all prospective scuba divers. Both the forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) should be at least 80% of predicted values and there should be no significant improvement (12% or more) with bronchodilators. An FEV_1/FVC ratio of less than 0.75 requires specialist opinion; however, the majority of people meeting these criteria simply have large vital capacities and are fit to dive.

Routine bronchial provocation testing is not needed. Exercise testing with spirometry up to 30 minutes after exercise should be performed if there is a history of exercise-induced wheeze.

Chest X-rays are not required for recreational scuba divers but should be performed in occupational divers and if there is any history of significant respiratory illness or any abnormal physical findings in recreational divers.

If there is doubt as to fitness to scuba dive, the standards require referral for specialist opinion.

Guide to pulmonary function testing and thoracic imaging

Pulmonary function tests

Introduction

Standard tests of pulmonary function, not all of which are available in Fiji, include spirometry, bronchial provocation testing and static lung volumes. Tests of gas exchange and gas transfer include analysis of arterial blood gases, pulse oximetry and diffusing capacity for carbon monoxide (DLCO). At the time of publishing this guideline, tests available in Fiji include arterial blood gases, pulse oximetry and spirometry.

Role of pulmonary function testing

Pulmonary function tests are useful for:

- assessing causes of breathlessness
- diagnosing respiratory conditions including
  - airway obstruction
  - interstitial lung disease
  - pulmonary vascular disorders
  - respiratory muscle weakness
  - disorders of ventilatory control
- assessing and monitoring asthma and other chronic chest disorders
- monitoring response to treatment
- assessing fitness to undergo surgery, to fly, to scuba dive and to enter certain occupations (eg the defence forces).

Contraindications to performing lung function testing include:

- recent cardiothoracic, intracranial, ear, nose, throat or ophthalmic surgery
- recent myocardial infarction, stroke, untreated pulmonary embolism, vascular aneurysm
- current pneumothorax or chest wall injury (with uncontrolled pain).

The patient must have good comprehension, the ability to cooperate with the testing, and be able to maintain a good lip seal around a mouthpiece for spirometry.

Spirometry and flow–volume loops

Spirometry is currently available at divisional hospitals, some sub-divisional hospitals, and some private health facilities.

Overview

Spirometry is the most commonly performed lung function test and perhaps the most useful. The testing equipment is relatively inexpensive, can be portable and is suitable to be used in a physician's office. Capacity for accurate and reproducible results depends on:

- quality equipment that can be calibrated and is well maintained
- operator technique
- the ability of the patient to follow instructions and give consistent effort; the test requires considerable physical effort and sick patients cannot perform it well.

Not all children can adequately perform lung function tests; success and reliability depends on various factors, including the child's age, the equipment and the operator. Extra time is generally needed to gain the confidence of younger children, and patience is required to obtain reliable results.

Spirometry is used to measure flow and volume during a forced expiratory manoeuvre, in which the patient first inhales to total lung capacity (TLC) and then exhales with maximum effort to residual volume (RV). Airflow measured at the mouth during this manoeuvre depends on lung elastic recoil, respiratory muscle strength and airway calibre, as well as patient cooperation in performing the test.

In the past, lung function equipment measured expired volume against time (a spirogram; see figure below). Most equipment now directly measures airflow as a function of volume and records this in a flow–volume loop (see figure below). It can also express the data as volume versus time. A maximum inspiratory flow volume manoeuvre is usually only performed in a specialist laboratory or if upper airway obstruction is suspected.

The common parameters measured with spirometry are:

- forced vital capacity (FVC)—the total volume exhaled
- forced expiratory volume in 1 second (FEV_1)—the volume exhaled in the first second
- FEV_1/FVC—expressed as a percentage or ratio (eg 70% or 0.7); may be referred to as forced expiratory ratio (FER).

Interpretation of spirometry

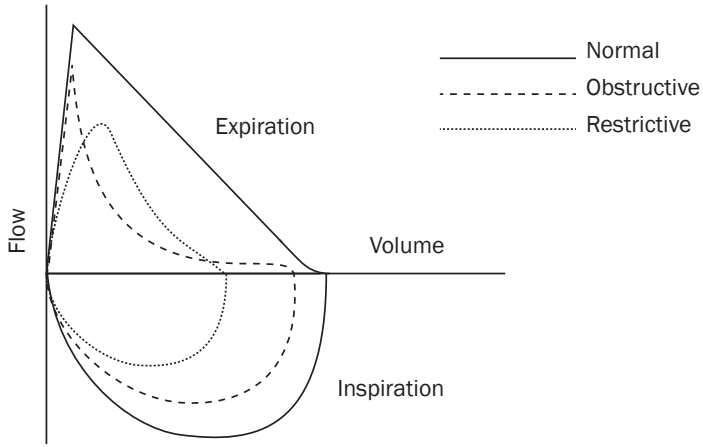
Overview

Spirometry results for an individual are compared with reference values matched for age, sex, height and ethnicity.

In the flow–volume loop of a healthy person, expiration results in a straight-sided triangle in the upper half of the loop, and inspiration produces a semicircle in the lower half (see the normal curve in the figure below).

The reference range for lung function values is approximately 80 to 120% of predicted. Spirometry is a robust test when done in accordance with established standards. There will be some individual variability on a day-to-day basis, but this is generally less than 10% (occasionally higher).

Figure 7: Flow–volume loops showing normal, obstructive and restrictive patterns



Reduced FEV_1 with FEV_1/FVC below the predicted range

A reduced forced expiratory volume in 1 second (FEV_1) with FEV_1/ forced vital capacity (FVC) below the predicted range indicates an obstructive ventilatory defect. Conditions causing airway obstruction include asthma and chronic obstructive pulmonary disease (COPD). FEV_1 is reduced but FVC is usually reduced to a lesser extent and, as a result, the ratio of FEV_1 to FVC is also reduced.

Reversible airflow limitation (bronchodilator response) is demonstrated by performing spirometry before and about 15 minutes after giving a bronchodilator (at least

200 micrograms of salbutamol). Current guidelines define a positive bronchodilator response as an increase above pre-bronchodilator baseline in FEV_1 and/or an increase in FVC of at least 12% and 200 mL.An increase of more than 12% and 400 mL is suggestive of asthma.

Reduced FEV_1 with normal or increased FEV_1/FVC

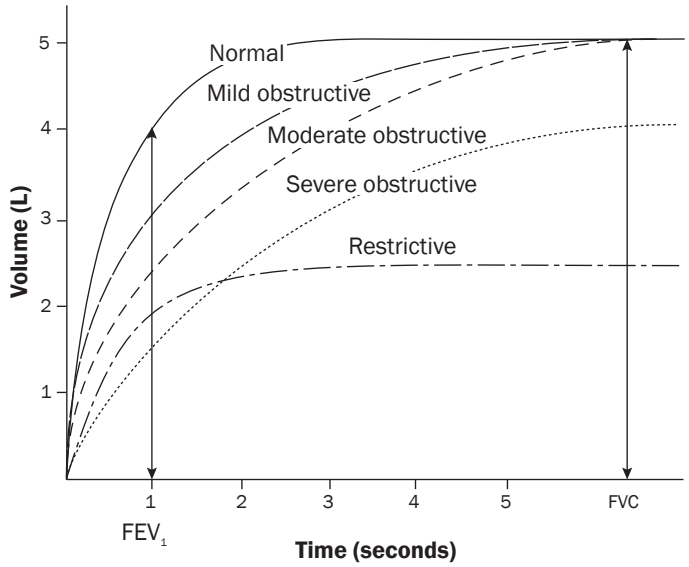
A reduced FEV_1 with normal or increased FEV_1/FVC indicates a **restrictive ventilatory defect**. Conditions causing a restrictive ventilatory defect include interstitial lung disease, respiratory muscle weakness and restrictive chest wall disease such as kyphoscoliosis. Both FEV_1 and FVC are reduced and the FEV_1/FVC remains normal.

Table 32: Classification of ventilatory defects by spirometry

	Obstructive	Restrictive
FEV_1	decreased	decreased
FVC	normal (or decreased if very severe)	decreased
FEV_1/FVC	decreased	normal

FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity

Figure 8: Spirograms showing normal, obstructive and restrictive patterns



FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity

Peak expiratory flow

Peak expiratory flow (PEF) measurements are useful in monitoring airway obstruction. PEF meters are usually small handheld devices for personal use. PEF measurements should always be performed on the same meter, as there is substantial variability in the results from different PEF meters.

PEF monitoring can be valuable in assessing the diurnal variability of airway obstruction (a characteristic feature of asthma), as well as the response to therapy. Measuring PEF may also be useful in the diagnosis and evaluation of occupational asthma.

The PEF technique is simple, with flow measured in Litres per minute (L/min). However, as an isolated measurement it is not a sensitive diagnostic tool and is less sensitive than forced expiratory volume in 1 second (FEV_1) when airway obstruction becomes severe. In general, PEF measurements are unreliable in children.

Bronchial provocation testing

Currently not available in Fiji.

Bronchial provocation testing identifies airway hyperresponsiveness, which is an exaggerated response to a bronchoconstrictor stimulus and is characteristic of asthma. The presence and severity of bronchial hyperresponsiveness may be used to assist in diagnosis or to follow the response to treatment.

Bronchial provocation tests should only be performed by experienced operators using standard laboratory protocols. The stimuli used include histamine and methacholine (direct tests), and cold dry air, nonisotonic aerosols and mannitol (indirect tests).

Tests of gas exchange and gas transfer

Arterial blood gas analysis

Currently available in divisional hospitals and some private health facilities.

Arterial blood gas analysis provides information to assess the blood oxygenation, acid–base balance and adequacy of ventilation by measuring:

- partial pressure of oxygen (PaO2) and carbon dioxide (PaCO2)
- pH
- values for bicarbonate concentration and base excess in a blood sample.

In adults, an arterial sample is usually taken from the radial or brachial artery, preferably under local anaesthetic. Collect the sample in a heparinised syringe, put on ice immediately, and analyse within 10 minutes.

Arterial blood gas analysis is indicated in:

- management of acute respiratory disorders requiring admission to hospital
- assessment of disorders associated with chronic ventilatory failure (ie with elevated PaCO2 levels)
- assessment for long-term domiciliary oxygen therapy.

Normal values for the parameters measured in arterial blood gas analysis are given in the table below. A guide to acid–base changes due to different pathophysiological states is provided in the next table.

Table 33: Normal values for arterial blood gas analysis

Parameter	Normal value
pH	7.40 plus or minus 0.04
PaO_2	higher than 85 mmHg breathing air values reduce with age; normal PaO_2 = 100 minus (0.2 × age)
$PaCO_2$	40 plus or minus 4 mmHg

$PaCO_2$ = partial pressure of carbon dioxide; PaO_2 = partial pressure of oxygen

Table 34: Guide to interpreting acid–base status from arterial blood gas analysis

Clinical state	Example	pH	$PaCO_2$	Bicarbonate concentration
Normal	—	7.4 plus or minus 0.04	40 plus or minus 4 mmHg	22 to 28 mmol/L
acute respiratory acidosis	hypercapnic COPD	decreased	increased	slight increase: 1 mmol/L for every 10 mmHg increase in $PaCO_2$
chronic respiratory acidosis	COPD	lower limit of normal	increased	increased
acute respiratory alkalosis	hyperventilation	increased	decreased	slightly decreased

cont...

chronic respiratory alkalosis	heart failure	upper limit of normal	decreased	decreased
acute metabolic acidosis	DKA	decreased	decreased	decreased
chronic metabolic acidosis	chronic kidney failure	mildly decreased	decreased	decreased
acute metabolic alkalosis	drug-induced, nasogastric tube	increased	increased	increased
chronic metabolic alkalosis	chronic vomiting and/or chronic diarrhoea	mildly increased	increased	increased

COPD = chronic obstructive pulmonary disease; DKA = diabetic ketoacidosis; $PaCO_2$ = partial pressure of carbon dioxide

Venous blood gas analysis

The use of venous blood gas samples to measure and follow partial pressure of carbon dioxide ($PaCO_2$), pH and bicarbonate concentration values has been promoted as an alternative to arterial blood gas samples. This has mainly come from emergency department and critical care literature.

Because venous blood gas values do not correlate directly with arterial blood gas values, there is considerable controversy as to whether venous blood gas analysis is clinically useful.

Pulse oximetry

Currently available at divisional and sub-divisional hospitals, some health centres and private health facilities.

Oxyhaemoglobin saturation (SaO_2) can be measured in clinical practice with a pulse oximeter (referred to as SpO_2). Pulse oximetry involves applying a close-fitting cap or clip containing light-emitting diodes to the finger or earlobe, which compare light absorption by oxyhaemoglobin and reduced haemoglobin. The method is noninvasive and inexpensive. It has become widely used in the care of patients with unstable respiratory disease or when close monitoring of oxygenation is needed.

Factors that can affect the accuracy of pulse oximetry include:

- poor circulation due to reduced cardiac output or peripheral vasoconstriction
- carboxyhaemoglobin or methaemoglobin—most available pulse oximeters

overestimate the SaO2 in the presence of even quite high concentrations of carboxyhaemoglobin or methaemoglobin. Causes of carboxyhaemoglobin include smoking, smoke inhalation, haemolysis, large haematoma; causes of methaemoglobin include dapsone, nitroprusside
- poor technique in the application of the monitoring device to the finger or earlobe
- faulty equipment
- some nail polish colours—placing the pulse oximeter sideways on the finger may improve accuracy
- dark skin.

In patients receiving supplemental oxygen, a normal SpO2 may be maintained despite worsening lung function and the development of hypercapnia and respiratory acidosis. If there is concern about the patient's condition (eg reduced level of consciousness), obtain arterial blood gas analysis with direct measurement of pH, partial pressure of oxygen (PaO2) and partial pressure of carbon dioxide (PaCO2).

Diffusing capacity for carbon monoxide

Currently not available in Fiji.

The diffusing capacity for carbon monoxide (DLCO) is a measure of the capacity of the lung to transfer gas from alveolar spaces into pulmonary capillary blood, ie the gas exchanging capability of the lung.

DLCO may be reduced in:
- interstitial lung disease
- emphysema
- pulmonary vascular disease
- anaemia (corrected for haemoglobin in most laboratories)
- drug-induced pulmonary toxicity.

Thoracic imaging

Introduction

The diagnosis of most respiratory problems can be clarified using spirometry and plain chest X-ray imaging. If further imaging is required specialist referral should be considered.

Chest X-ray

Currently available in divisional and sub-divisional hospitals, some health centres and some private health facilities.

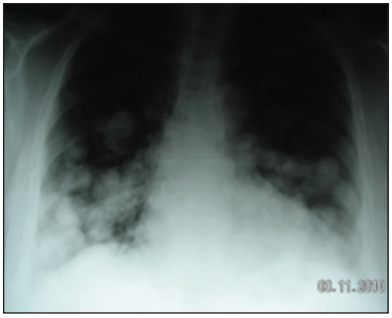
Chest X-ray is a useful investigation when lung pathology is suspected and should be performed before requesting other imaging. The dose of radiation involved is very low—0.02 to 0.1 mSv depending on the number of views taken (eg just posterior–anterior [PA] or also with a lateral view). This dose is equivalent to 4 to 18 days of background radiation.

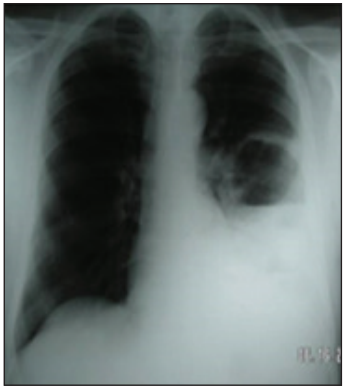
For assessment in primary care, a chest X-ray can:

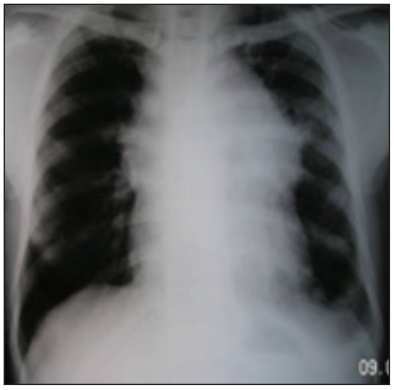
- distinguish pneumonia from bronchitis
- diagnose important causes of breathlessness such as pneumothorax, pleural effusion, pneumonia, interstitial lung disease or cardiac failure
- identify lung mass(es) in a patient presenting with symptoms suggesting malignancy.

Compare chest X-rays to the patient's previous X-rays when possible. In some situations a follow-up chest X-ray should be done at an appropriate interval (eg to follow pneumonia to resolution, particularly in a smoker), which depends on the possible pathology.

Images 10.1, 10.2 and 10.3: Examples of chest X-ray images







Computed tomography of the chest

Currently available in divisional hospitals and some private health facilities.

General considerations

Computed tomography (CT) of the chest is the key to diagnosis of many lung diseases; however, many requests for CT of the chest, both in general and hospital practice, lack clinical justification and are therefore inappropriate.

CT of the chest is expensive and results in high radiation exposure. The effective dose varies between different machines, and also the patient size, but is typically around 8 mSv, which is equivalent to 4 years of background radiation or 400 chest X-rays. The dose to the breast may be as high as 10 to 33 mSv. The International Commission on Radiological Protection estimates that this dose will cause a fatal cancer in 1 in 2500 people exposed overall; the risk is higher in younger people and females.

CT scanning of the chest can be performed as conventional CT chest with contrast injection, high-resolution CT (HRCT) scanning or CT pulmonary angiography (CTPA).

Conventional CT chest with contrast injection

Conventional CT chest with contrast injection is the modality of choice to image the chest wall, mediastinum and pleura, and for evaluation of lung masses. It is usually the first staging investigation for lung cancer, when it is combined with upper abdomen CT to detect liver and adrenal metastases. However, if there is a clinically obvious pulmonary mass or pleural effusion, the first step should be definitive histological diagnosis before CT is requested.

High-resolution CT

In high-resolution CT (HRCT) scanning, 1 mm cuts of the lungs are examined at intervals of 7 to 10 mm. It is used in the diagnosis and assessment of bronchiectasis and interstitial lung diseases. Only a small fraction of the lung volume is examined by the radiologist and small focal lung lesions can be missed. The effective dose from an HRCT scan is about 2 to 8 mSv depending on the technique used.

CT pulmonary angiography

CT pulmonary angiography (CTPA) has become the most common investigation for suspected pulmonary embolism. A drawback is the high radiation exposure, especially to the breast.

CTPA should not be used to screen for pulmonary embolism when clinical probability of pulmonary embolism is low; tests for venous thrombosis are preferred in this group. If screening investigations are positive or clinical suspicion is high, CTPA may be appropriate.

CTPA may be contraindicated for patients with allergies to contrast media, kidney disease or diabetic nephropathy. The effective dose from a CTPA scan is about 7 to 10 mSv.

Ultrasound of the thorax

Ultrasound of the thorax is the most sensitive method for detecting pleural effusions and for demonstrating loculations in pleural effusions. Ultrasound can be used at the bedside for guiding diagnostic aspiration or insertion of intercostal catheters.

Inhalational drug delivery devices

General principles

Introduction

Many respiratory drugs are delivered directly to the airway by inhalational devices. This achieves a higher drug concentration at the receptors and a more rapid onset of action than systemic delivery, resulting in fewer systemic adverse effects.

There are a growing number of different inhalational delivery devices available, which can cause confusion for both patients and practitioners. At the time of writing, inhalational delivery devices available in Fiji for managing asthma and chronic obstructive pulmonary disease (COPD) can be categorised as:

- pressurised metered dose inhalers (MDIs)
- nebulisers.

Inhaler technique

Repeated assessment and demonstration of correct inhaler technique are essential. Up to 90% of patients use an incorrect technique with their device, resulting in inadequate drug delivery to the lungs. Check patient technique and demonstrate the correct technique, if necessary, at every opportunity. This is important because patients are often unaware they are using the devices incorrectly, and their technique can deteriorate over time.

Check inhaler technique at each review and demonstrate correct technique if required.

Advice on device-specific technique, including videos is available from <www.nationalasthma.org.au/how-to-videos/using-your-inhaler>

Also, refer to the sample Asthma Action Plan for adults and adolescents (pages 210-211) for instruction to inhaler technique.

Pressurised metered dose inhalers

Pressurised metered dose inhalers (MDIs) are multiple-dose drug delivery devices, usually containing a propellant system such as hydrofluoroalkane

The closed-mouth technique is the only approved method for use of a MDI without a spacer. The lips are sealed around the mouthpiece of the MDI after exhalation

and before the inhaler is actuated. The open-mouth technique, where the inhaler is actuated when held up to 6 cm away from the open mouth, is no longer recommended.

Patients with arthritis or weakened hand muscles may have difficulty pressing down on the MDI canister. Using two hands to actuate the inhaler may be easier. A plastic lever device (Haleraid) can be attached to some MDIs to increase pressure. Currently, they are not readily available in Fiji, but could be ordered from community pharmacies.

For some MDIs correct cleaning is particularly important to prevent blockage and to ensure delivery of the intended dose. Cleaning instructions vary for the different devices; consult product information. However, in general:

- corticosteroid-containing inhalers—never wash
- other MDIs—wash weekly.

Spacer devices

Spacers hold the aerosol cloud from the MDI in a confined space sealed with a valve system which allows subsequent inhalation. Using a spacer device with pressurised metered dose inhalers (MDIs) is essential for children and recommended for adults.

In the spacer, evaporation of some of the propellant produces particles of smaller size, which may increase endobronchial drug deposition and decrease oropharyngeal drug deposition. For inhaled corticosteroids, this reduces oropharyngeal candidiasis, dysphonia and systemic absorption.

In most circumstances, a MDI with spacer should be used instead of a nebuliser. This includes in first aid for asthma flare-ups, and in other emergency situations. However, for treatment of life-threatening asthma, or if forced expiratory volume in 1 second (FEV_1) is less than 30% predicted, use of a nebuliser is recommended.

Spacer devices may be small volume, large volume, or cardboard, which is only recommended for single-patient short-term use (ie up to 7 days). Smaller spacers are now available for adults.

Different spacers have different priming and cleaning requirements, depending on the material they are made from and its propensity for forming electrostatic surface charge. Unlike the plastic spacers commonly used in the past (eg Volumatic), polyurethane spacers may not require washing or priming before first use. Disposable cardboard spacers do not require priming or washing.

It is best to wash a new plastic spacer before first use to reduce electrostatic charges. If it must be used immediately, the static charge may be reduced by actuating the inhaler multiple times into the spacer. However, the optimal number of puffs for priming is unknown; the findings from in vitro studies vary widely.

Plastic and polyurethane spacers require regular washing with water and mild detergent (without rinsing). Air dry the spacer; do not use a cloth to dry it. See the manufacturers' instructions for further details, including when to replace the spacer.

Most spacers have pliable fittings, so they are compatible with all MDIs. Adapters may be needed for spacers with fixed fittings; these are available from pharmacies or the inhaler manufacturer.

A face mask adapter (available from pharmacies) may be used with some spacers for infants, young children, and patients unable to form a lip seal around the spacer mouthpiece (eg people with cognitive impairment or disabilities).

Spacers should not be used by multiple patients except in emergencies or if the manufacturer specifies they can be autoclaved.

Nebulisers

Use of nebulisers is decreasing because an equivalent bronchodilator effect can be achieved by inhalation via a spacer using a short-acting beta$_2$ agonist pressurised metered dose inhaler (MDI). However, for life-threatening asthma, or if forced expiratory volume in 1 second (FEV_1) is less than 30% predicted, use of a nebuliser is recommended.

Nebulisers produce a respirable aerosol from a drug solution. Jet nebulisation can use compressed air or oxygen to drive the system. Oxygen is only used to drive nebulisation for patients with acute life-threatening asthma flare-ups. Oxygen should not be used to drive nebulisation for patients with acute exacerbations of chronic obstructive pulmonary disease (COPD).

Images 11.1 and 11.2: Examples of nebuliser masks

Inhalational drug delivery devices and device-specific considerations

Table 35: Inhalational drug delivery devices and device-specific considerations

Device type	Advantages	Precautions
Drug delivery devices		
Pressurised metered dose inhaler	• compatible with spacers to improve lung deposition of drug	• require good hand–breath coordination, especially if not used with a spacer • require sufficient hand strength to actuate—a plastic lever device (Haleraid) is available for some MDIs to assist; for other MDIs, suggest using two hands to actuate • unsuitable for use without a spacer in children younger than 8 years old • not all products have dose counters • some devices need to be primed before first use or if not used for several days (see product information for details)
Nebuliser	• useful in some acute situations, eg v less than 30% predicted	• higher doses of drug delivered, so systemic adverse effects may be greater • expensive • longer time required to deliver equivalent dose compared to other devices • regular device servicing needed • not suitable for use in patients with acute infection due to potential to spread infective organisms • risk of acute angle glaucoma if using antimuscarinic drugs • risk of local adverse effects (eg to face) if using mask rather than mouthpiece when nebulising corticosteroids

cont...

Spacer devices		
small volume	• recommended for use with MDI containing ICS and any MDI used by children • reduced oropharyngeal deposition—reduction in local adverse effects • increased lung deposition • useful for people with poor hand–breath coordination	• device must be compatible with the spacer; spacers either have an oval or round fitting • for plastic spacers, washing with detergent required before first use (without rinsing) to reduce electrostatic adhesion • cleaning required weekly to monthly depending on device (see product information), and after the resolution of a respiratory tract infection. Cleaning plastic spacers too frequently can increase electrostatic charge
large volume		
Disposable	• collapsible; useful when travelling	• to be used by one patient for up to 7 days only
face mask (with spacer)	• useful for – small children – people with cognitive impairment – people with disabilities – people unable to form a lip seal with a spacer	

FEV_1 = forced expiratory volume in 1 second; ICS = inhaled corticosteroids; MDI = pressurised metered dose inhaler

Respiratory therapy in pregnancy and breastfeeding

Pregnancy and respiratory drugs

There is no convincing evidence that any of the drugs commonly used to treat respiratory disorders cause particular problems during pregnancy. As a general principle, the lowest dose achieving best control should be used. Inhalation has particular advantages as a means of drug administration during pregnancy. The therapeutic effect may be achieved without reaching plasma concentrations likely to have a pharmacological effect on the fetus.

The major period of danger for teratogenic effects of any drug is the first trimester of pregnancy, although some drugs can interfere with functional development of organ systems and the central nervous system in the second and third trimesters.

Breastfeeding and respiratory drugs

The benefits of breastfeeding are sufficiently important to recommend that breastfeeding should be continued unless there is substantial evidence that the drug taken by the mother will be harmful to the infant and that no therapeutic equivalent can be given.

Most drugs are excreted only to a minimal extent in breastmilk and in most cases the dosage to which the infant is ultimately exposed is very low and is well below the therapeutic dose level for infants. In most situations, drugs cross the placenta more efficiently than they pass into breastmilk.

Inhalation has particular advantages as a means of maternal drug administration during breastfeeding because the therapeutic effect may be achieved without reaching plasma concentrations that may contribute to the drug entering breastmilk.

The table below provides advice on the safety of individual drugs used in respiratory disorders in breastfeeding women.

Table 36: Respiratory drugs in pregnancy and breastfeeding

Drug	**pregnancy category**	**Compatibility with breastfeeding** [Note 1]
adrenaline (epinephrine)	Safe to use	Compatible
aminophylline	Ideally, plasma theophylline concentration should be monitored as pharmacokinetics (and dose requirement) may change. However, this is currently not available in Fiji.	Compatible but irritability has been reported in infants; monitor infant and keep dose as low as possible. Inhaled bronchodilators preferred.
azelastine (intranasal) Non-EML	Limited information; alternatives preferred.	Compatible
beclomethasone (inhaled)	Safe to use	Compatible
budesonide (inhaled) Non-EML	Safe to use	Compatible
cetirizine Non-EML	Safe to use	Compatible
cromoglycate (inhaled, intranasal) Non-EML	Safe to use	Compatible
desloratadine Non-EML	Consider alternative, there is more experience with other antistamines	Compatible
dexamethasone	Safe to use, although transfer across the placenta is greater than with hydrocortisone and prednislone. Use the lowest effective dose for the shortest possible time.	Use with caution
fexofenadine Non-EML	Consider alternative, there is more experience with other antistamines	Compatible

cont...

Drug	pregnancy category	Compatibility with breastfeeding [Note 1]
formoterol (eformoterol) Non-EML	Limited information available, continue formoterol if part of pre-pregnancy asthma management or if considered the medicine of choice.	Compatible
fluticasone propionate Non-EML	Safe to use	Compatible
hydrocortisone	Safe to use. Use the lowest effective dose for the shortest possible time.	Compatible
ipratropium (inhaled)	Limited information; considered safe to use.	Compatible
levocabastine (intranasal, eye drop) Non-EML	Safe to use	Compatible
lignocaine	Safe to use	Compatible
loratadine Non-EML	Safe to use, more data with loratadine than other non-sedating antihistamines.	Compatible
magnesium sulfate	Safe to use	Compatible
methylprednisolone	Safe to use. Use the lowest effective dose for the shortest possible time.	Compatible; caution with high doses, consider withholding breastfeeding for 4 hours after dose.
montelukast Non-EML	Limited information, considered safe to use.	Compatible.
prednisolone	Safe to use. Use the lowest effective dose for the shortest possible time.	Compatible
prednisone	Safe to use. Use the lowest effective dose for the shortest possible time	Compatible

cont...

Drug	pregnancy category	Compatibility with breastfeeding [Note 1]
promethazine hydrochloride	Safe to use; avoid close to term (theoretical risk of neurological disturbances in the newborn).	Compatible but less sedating antihistamines preferred; monitor the infant for irritability and sleep disturbances.
salbutamol	Safe to use; when used intravenously monitor both mother and foetus for increased heart rate and blood pressure reduction	Compatible
salmeterol Non-EML	Limited experience but considered safe to use; continue if part of pre-pregnancy asthma management or if considered the medicineof choice..	Compatible
terbutaline Non-EML	Safe to use	Compatible
theophylline	Continue if part of pre-pregnancy asthma management as poorly controlled asthma increased the risk of adverse pregnancy outcomes. Ideally, plasma theophylline concentration should be monitored as pharmacokinetics (and dose requirement) may change. However, this is currently not available in Fiji. The newborn should be monitored for signs of toxicity (eg tachycardia, irritability and vomiting) after delivery.	Compatible; monitor infant for irritability

cont...

Drug	pregnancy category	Compatibility with breastfeeding [Note 1]
tiotropium [Non-EML]	Limited information; continue if part of pre-pregnancy asthma or COPD management.	Compatible

Note 1: Definitions for compatibility with breastfeeding:

- compatible—there are sufficient data available to demonstrate an acceptably low relative infant dose and/or no significant plasma concentrations and/or no adverse effects in breastfed infants
- caution—there are insufficient data showing low relative infant dose and/or no significant plasma concentrations and/or no adverse effects in breastfed infants
- avoid, insufficient data—there are no data on transfer into milk, or on plasma concentrations or adverse effects in the breastfed infant
- avoid—significant plasma concentrations in exposed infants, or adverse effects in breastfed infants reported or predictable from the properties of the molecule.

[Non-EML] medication not included on the Fiji Essential Medicines List (EML) at the time of publication.

Appendices

Appendix 1: Asthma Action Plan for children > 5 years of age

My Asthma Action Plan

Age ≥5 years

Patient Name: _____

Medical Record #: _____

Clinician's Name: _____ DOB: _____

Clinician's Phone #: _____ Completed by: _____ Date: _____

Long-Term Control Medicines	How Much To Take	How Often	Other Instructions
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	
Quick-Relief Medicines	**How Much To Take**	**How Often**	**Other Instructions**
		Take ONLY as needed	NOTE: If this medicine is needed frequently, call clinician to consider increasing long-term control medications.

Special instructions when I feel good, not good, and awful.

GREEN ZONE

I feel *good*.

{My peak flow is in the GREEN zone.}

My Personal Best Peak Flow

80% Personal Best

50% Personal Best

Unable

Peak Flow Meter

PREVENT asthma symptoms everyday:

- ☐ Take my long-term control medicines (above) every day.
- ☐ Before exercise, take _____ puffs of _____
- ☐ Avoid things that make my asthma worse like:

_____

YELLOW ZONE

I do *not* feel *good*.

{My peak flow is in the YELLOW zone.}

My symptoms may include one or more of the following:

- Wheeze
- Tight chest
- Cough
- Shortness of breath
- Waking up at night with asthma symptoms
- Decreased ability to do usual activities
- _____

CAUTION. I should continue taking my long-term control asthma medicines every day AND:

- ☐ Take _____

If I still do not feel good, or my peak flow is not back in the *Green Zone* within one hour, then I should:

- ☐ Increase _____
- ☐ Add _____
- ☐ Call _____

RED ZONE

I feel *awful*.

{My peak flow is in the RED zone.}

Warning signs may include one or more of the following:

- It is getting harder and harder to breathe
- Unable to sleep or do usual activities because of trouble breathing

MEDICAL ALERT! Get help!

- ☐ Take _____ until I get help immediately.
- ☐ Take _____
- ☐ Call _____

Danger! Get help immediately!

Call for an ambulance OR rush to your nearest hospital if you have trouble walking or talking due to shortness of breath or lips or fingernails are gray or blue.

Appendix 2: Asthma Action Plan 1 to 5 years of age

Child Asthma Action Plan

1 to 5 years old

Patient Name: _____

Medical Record #: _____

Healthcare Provider's Name: _____

DOB: _____

Healthcare Provider's Phone #: _____

Completed by: _____ Date: _____

Long-Term Control Medicines (Use Every Day To Stay Healthy)	How Much To Take	How Often	Other Instructions (such as spacers/masks, nebulizers)
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	
		_____ times per day EVERY DAY!	

Quick-Relief Medicines	How Much To Take	How Often	Other Instructions
		Give ONLY as needed	NOTE: If this medicine is needed often (_____ times per week), call clinician.

GREEN ZONE

Child is ***well*** and has no asthma symptoms, even during active play.

PREVENT asthma symptoms everyday:

- Give the above long-term control medicines every day.
- Avoid things that make the child's asthma worse:

☑ Avoid tobacco smoke; ask people to smoke outside.

☐ _____

☐ _____

YELLOW ZONE

Child is ***not well*** and has asthma symptoms that may include:

- Coughing
- Wheezing
- Runny nose or other cold symptoms
- Breathing harder or faster
- Awakening due to coughing or difficulty breathing
- Playing less than usual
- _____
- _____

Other symptoms that could indicate that your child is having trouble breathing may include: difficulty feeding (grunting sounds, poor sucking), changes in sleep patterns, cranky and tired, decreased appetite.

CAUTION. Take action by continuing to give regular asthma medicines every day AND:

☐ Give _____ (include dose and frequency)

If the child is not in the ***Green Zone*** and still has symptoms after one hour then:

☐ Give more _____ (include dose and frequency)

☐ _____ (include dose and frequency)

☐ Call _____

RED ZONE

Child ***feels awful!*** Warning signs may include:

- Child's wheeze, cough, or difficulty breathing continues or worsens, even after giving yellow zone medicines.
- Child's breathing is so hard that he/she is having trouble walking/talking/eating/playing.
- Child is drowsy or less alert than normal.

Danger! Get help immediately!

MEDICAL ALERT! Get help!

☐ Take the child to the hospital or call 9-1-1 immediately!

☐ Give more _____ until you get help. (include dose and frequency)

☐ Give _____ (include dose and frequency)

Call for an ambulance OR RUSH TO YOUR NEAREST HOSPITAL IF:

- The child's skin is sucked in around neck and ribs, or
- Lips and/or fingernails are gray or blue, or
- Child does not respond to you

Appendix 3: Asthma Action Plan adults & adolescents

ASTHMA ACTION PLAN

Ministry of Health and Medical Services Fiji

Take this asthma action plan with you when you visit your doctor or health facility

Name: ..... Emergency contact (name/number): .....

Doctor, nurse, health provider: ..... Date of plan: .....

My trigger factors: .....

	Asthma severity	Other instructions	Preventer therapy	Reliever therapy
	 My best peak flow is: *The peak flow test measures how fast you can breathe out, so you can see how well your lungs are working.*	 *A spacer helps you to get the best from your medicine.* *Always use a spacer with your preventer and reliever puffers if available.*	 My preventer puffer is: *Your preventer medicine reduces swelling and mucus in the airways of your lungs. After using, rinse your mouth with water and spit out.*	 My reliever puffer is: *Your reliever medicine works quickly to make breathing easier by making the airways wider.* **Carry your reliever at all times!**
GREEN ZONE well-controlled	I am well (is all of these) • I have shortness of breath or cough occasionally • I do not wake at night with asthma • I can do all my usual activities • I can exercise My peak flow is more than ☐ *(> 80% of best peak flow)*	• Avoid trigger factors • If sport makes it hard to breathe, take **2** puffs of reliever inhaler 15 minutes before exercise	Take: ☐ puff(s) ☐ time(s) a day every day **even when well.** *Unless stopped by my doctor*	Take 2 puffs whenever you have asthma symptoms: • Wheezing • Chest feels tight • It's hard to breathe • Coughing
YELLOW ZONE flare up	I am not well (any of these) • I have shortness of breath or cough, or • I'm waking at night with asthma symptoms, or • I'm unable to do my usual activities, or • I require my reliever therapy more than 3 times per week, or My peak flow is less than ☐ *(< 80% of best peak flow)*	• See your doctor or nurse at your nearest health facility on the **same day** • Avoid trigger factors	Take: ☐ puff(s) **TWO** times a day every day even when well	Take **2** puffs **FOUR** times a day plus **2 PUFFS** whenever you have asthma symptoms: • Wheezing • Chest feels tight • It's hard to breathe • Coughing
ORANGE ZONE severe asthma	**I am very sick (any of these)** • **I'm finding it hard to breathe, or** • **I'm waking often at night with asthma symptoms, or** • **My reliever effect isn't lasting 3 hours, or** **My peak flow is less than ☐ or is falling over the day** *(< 60% of best peak flow)*	• If you have been given prednisolone (steroid) tablets **take a single dose of 60 mg straight away** • Go to the nearest health centre **immediately**	Take: ☐ puff(s) **TWO** times a day every day.	Take **2** puffs **FOUR** times a day plus whenever you have asthma symptoms: • Wheezing • Chest feels tight • It's hard to breathe • Coughing !
				If asthma is not relieved, start first aid (opposite page)
RED ZONE emergency	Emergency is any of these danger signs: • I'm finding it very hard to breathe • I can't speak a full sentence • My reliever puffer is not helping • I feel like my asthma is out of control • I'm feeling terrified • I'm feeling exhausted	!	Start asthma first aid – turn over page Seek medical help immediately	

ASTHMA FIRST AID

Ministry of Health and Medical Services Fiji

How do I know someone is having a severe asthma attack? Look out for these danger signs:

If someone has any one of these danger signs start asthma first aid straight away and seek medical help immediately.

4 steps of asthma first aid

1

Sit the person upright
- Be calm and reassuring
- Do not leave the person alone
- Call out for assistance

2

Give **4 separate puffs** of (blue/grey) reliever puffer
- Shake puffer
- Put **1 puff** into spacer
- Take **4 breaths** from spacer
- Repeat until all 4 puffs have been taken

Remember: shake puffer, 1 puff, 4 breaths
If no spacer available, take **1 puff** as you take a slow deep breath. Repeat until all **4 puffs** given.

3

Wait **4 minutes**

If the person still cannot breathe easily, give **4 more puffs** as above

4

- Call an ambulance – say that the person is having a '**severe asthma attack**' – or help the person get to the nearest health facility immediately.
- **Continue giving 4 puffs of the reliever puffer every 4 minutes**

How to use asthma puffers

With spacer

1. Remove the cap and shake puffer well
2. Insert puffer into spacer
3. Tilt head back slightly
4. Place mouthpiece between teeth and seal lips around it
5. Press once firmly on puffer to release one puff into spacer
6. Take 4 breaths in and out of spacer
7. Repeat 1 puff at a time – shake puffer between each puff
8. Replace puffer cap

Without spacer

1. Remove the cap and shake puffer well
2. Breathe out away from puffer
3. Tilt head back slightly
4. Place mouthpiece between teeth and seal lips around it
5. Press once firmly on puffer at the same time breathing in slowly and deeply
6. Slip puffer out of mouth
7. Hold breath for 5 seconds or as long as is comfortable
8. Repeat 1 puff at a time – shake puffer between each puff
9. Replace puffer cap

Carry your reliever puffer with you at all times
Ask your doctor, pharmacist or other healthcare provider if you are not sure how to use your puffer.