RESPIRATORY GUIDELINES

Medicines Used in Respiratory Diseases

Introduction

This chapter contains brief summaries of the major drugs used in the management of respiratory disease and are recommended in these guidelines. The summaries do not contain comprehensive accounts of the pharmacology of these compounds. The reader is advised to consult standard textbooks and/or the industry product information for more details.

It is important to consider the risks and benefits of drugs (particularly corticosteroids) that are used to treat respiratory diseases. As a general principle, the lowest drug doses that achieve best control should be used. For example:

Patient adherence to asthma treatment is better if regimens have:

fewer devices and drugs
fewer adverse effects
been understood and agreed between patient and health care
professional.

Beta₂ receptor stimulating drugs (Beta₂ agonists) Introduction

Stimulation of beta₂-receptors on airway smooth muscle relaxes the muscle resulting in bronchodilation. All beta₂ agonists may also stimulate beta₁-receptors; however, the effects of beta₁-receptor stimulation (eg tachycardia) are more likely to occur following systemic absorption or following inhalation of relatively large doses.

Under almost all circumstances, the preferred route of administration for beta₂ agonists is by inhalation. Administration by inhalation causes bronchodilation with low doses thus minimising systemic adverse effects.

Adverse effects: Dose-limiting adverse effects of the beta₂ agonists are most commonly tachycardia (which can also lead to paroxysmal tachyarrhythmias, such as atrial fibrillation or paroxysmal supraventricular tachycardia), tremor, headaches, muscle cramps, insomnia, and a feeling of anxiety and nervousness. In high doses (eg tablets,intravenous and emergency nebulisation) all beta₂ agonists can cause hypokalaemia and hyperglycaemia.

Short-acting beta2 agonists

Salbutamol

Salbutamol is the only drug in this class in the Tuvalu Essential Medicine List (EML) and is available in puffers, inhalation solution, 2mg/5mL oral solution and 4mg tablets. It is a fast-acting bronchodilator; the effects are evident within 5 minutes and last for about 3 hours. It is used to relieve bronchoconstriction and is often referred to as a **reliever** (**blue puffer**) medication.

Precautions: Regular and frequent use of salbutamol without appropriate attention to other aspects of respiratory illness is inadvisable. Proper consideration of issues such as an asthma management plan, monitoring of symptoms and lung function and suitable and preventive therapy are important when salbutamol is used in the regular management of airways disease. In general, it should be reserved for intermittent symptom relief rather than regular treatment of asthma. High-volume, regular use may indicate that the underlying disease process is poorly controlled, warranting modification of other aspects of drug therapy. The use of one or more canisters per month is associated with a greater risk of hospital admission.

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Anticholinergic bronchodilators

Ipratropium bromide

Ipratropium bromide is the drug of this type which is not available in the Tuvalu Essential Medicine List (EML) and can be given by inhalation. It is a short-acting anticholinergic drug that produces bronchodilation by blocking vagal tone and reflexes, which mediate bronchoconstriction.

Used alone, ipratropium bromide is not a powerful bronchodilator. The duration of action is approximately 6 hours. Although the onset of action is 3 to 5 minutes, peak effect is not reached until 1.5 to 2 hours, so ipratropium bromide should not be used for immediate relief of symptoms. The drug can be used to augment the duration of bronchodilation achieved with beta₂ agonist therapy and is recommended for acute severe asthma and in chronic obstructive pulmonary disease (COPD).

Adverse effects related to the anticholinergic action of ipratropium bromide are uncommon, as this drug is poorly absorbed. Some local adverse effects, such as blurred vision or precipitation of glaucoma in susceptible individuals, may result from inadvertent contact of nebulised drug with the eyes. Similarly, although buccal absorption is slight, some patients may experience a dry mouth. Systemic anticholinergic effects are very rare.

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Xanthines (Theophylline and Aminophylline)

Although the mechanism of action is not well understood, xanthines may relax smooth muscle and increase diaphragm contractility. The xanthine agents available in the Tuvalu Essential Medicine List (EML). Aminophylline, a derivative of theophylline, administered intravenously. In routine use, the bronchodilator actions of theophylline offer no advantage over beta₂ agonists. Aminophylline should be reserved for severe acute asthma failing to respond to standard management. For patients not taking theophylline a bolus loading dose should be given. This must be given slowly over 5-10 minutes or severe side effects will result.

The efficacy of theophylline is difficult to demonstrate in patients with COPD; however, it may be helpful in some individuals. Theophylline should be considered only for patients in whom other treatment has failed to control symptoms adequately (eg after a trial of short-acting bronchodilators and long-acting bronchodilators), or in patients who are unable to use inhaled therapy.

Adverse effects, interactions and precautions:

These drugs have a number of unpleasant side effects including nausea and vomiting, insomnia, cardiac arrhythmias, seizures, and hypokalaemia. The liver enzymes responsible for theophylline metabolism are inhibited by a range of drugs, including macrolide antibiotics (eg erythromycin), quinolone antibiotics (eg ciprofloxacin).. COPDministration of theophylline with these drugs may cause serious theophylline toxicity. In situations where such combination therapy cannot be avoided, the patient must be monitored closely.

The enzymes responsible for the metabolism of the ophylline may also be induced by ciagarette smoking and by other drugs, including rifampicin, and some anticonvulsants (eg phenytoin, carbamazepine, barbiturates). The introduction of concurrent therapy with one of these drugs may result in a loss of the therapeutic effect of the ophylline.

Theophylline has a narrow therapeutic window, and the dosage for maintenance therapy should be based on assessment of clinical response and adverse effects Lower doses may be required in the elderly or in hepatic impairment. Smokers will usually need higher doses. In the management of COPD, satisfactory clinical response may be attained with lower doses than are necessary for the treatment of asthma. Theophylline is well absorbed after oral administration, and has a highly variable half-life of approximately 8 hours in adults and 4 hours in children.

Corticosteroids

Introduction

Corticosteroids are widely used in the treatment of asthma and other respiratory diseases to reduce bronchial inflammation and hyperresponsiveness. They are thought to reduce the synthesis and secretion of a variety of inflammatory mediators (such as

prostaglandins and leukotrienes) and cytokines, which are implicated in the pathogenic process underlying asthma.

Corticosteroids are used in the management of both acute severe asthma and the preventive management of asthma. The agent available on the Tuvalu Essential Medicine List (EML) are prednisolone and is given orally, hydrocortisone and dexamethasone (used parenterally) and beclomethasone diproprionate given byinhalation..

Note that dexamethasone oral formulation is not available on the Tuvalu EML but is effectively interchangeable with prednisolone.

Inhaled corticosteroids

Beclomethasone dipropionate, is used as preventive (brown puffer) therapy in asthma. It has a delayed onset of clinical effect and should be used regularly. It is not sufficiently potent and does not have a sufficiently rapid effect to be of use in acute severe asthma.

Adverse effects, interactions and precautions: Inhaled corticosteroids do not generally produce systemic adverse effects until large doses are administered. Systemic effects are dependent on a complex interplay between:

□ potency of the corticosteroid
□ absorption of the drug deposited in the airway, and delivery device
used (MDI with or without spacer),
absorption of drug deposited in the pharynx and swallowed, and first-
pass hepatic metabolism (to a minor degree).

. In adults, doses at which systemic adverse effects may become manifest are those greater than 500 to 750 micrograms daily of beclomethasone In children, doses at which systemic adverse effects may become manifest are those greater than 400 micrograms daily. Systemic adverse effects occur at lower doses in some patients, and the possibility of cataracts should be considered, particularly in those receiving therapy of extended duration. The dose at which the hypothalamic-pituitary-adrenal (HPA) axis is suppressed has not yet been established for any corticosteroid, so the lowest effective dose should always be recommended.

The effect of inhaled corticosteroids on long-term growth in children is unclear. Most studies have focused on short-term growth velocity and have failed to show any reduction in final height. In fact, children with severe asthma may have improved growth velocity after starting inhaled corticosteroids, perhaps by eliminating the growth-suppressive effects of poorly controlled asthma.

In low doses adverse effects are uncommon, but include hoarse voice and oral and oesophageal *Candida albicans* infection (candidiasis/thrush). To minimise oropharyngeal thrush and absorption of inhaled corticosteroids, patients should be advised to rinse their throat and mouth with water and spit out after inhalation. Patients using a puffer hould also be encouraged to use a spacer

Corticosteroid nasal sprays may cause sneezing, nasal irritation and nosebleeds.

Oral corticosteroids

Oral prednisolone is well absorbed and is eliminated by liver metabolism. Its plasma half-life is approximately 3 hours; however, the biological action is prolonged for up to 24 hours. Its metabolism is enhanced by drugs that induce liver enzymes (eg phenytoin and carbamazepine) and inhibited by drugs that inhibit liver enzymes (eg erythromycin, roxythromycin).

Consideration of the patient's weight and age, as well as the severity of the disease being treated, should guide the dosage regimen for systemic corticosteroids. In general the lowest dose possible to achieve the desired clinical response should be used. Prednisolone is usually given as a short course lasting several days to weeks with the aim of disease control without exposing the patient to the corticosteroid for a long enough period for significant adverse effects to develop..

It is generally given as a single daily dose in the morning to mimic the natural cortisol peak. Dosing in the evening often results in sleep disturbances.

Dose reduction (tapering)

The hypothalamic-pituitary-adrenal axis is suppressed by glucocorticoid therapy. The dose, duration of treatment and individual patient characteristics affect the onset and extent of this effect. However, treatment with prednisolone at doses of 5 -10mg for longer than 2 weeks can be sufficient to cause adrenal suppression. Therefore tapering of the dose is required to avoid both adrenal insufficiency and also rebound in symptoms, which may occur with sudden cessation. The rate of reduction is dependent on the dose level, duration of treatment and underlying disease state.

Adverse effects

Systemic corticosteroid treatment inevitably results in adverse effects if the dose and/or duration of treatment are sufficient, because most are dose-related biological effects of the hormone.

Table 6. Important complications of corticosteroids

Glucocorticoid	Mineralocorticoid	
☐ gastrointestinal effects (eg dyspepsia,	☐ hypertension	
risk	☐ hypokalaemic alkalosis	
factor for peptic ulceration, gastrointestinal bleeding)	☐ sodium-retaining effects	
☐ growth retardation		
☐ immunosuppression, risk of infections		
☐ metabolic effects (eg diabetes, hypertriglyceridaemia)		
☐ myopathy		
ocular effects, particularly increased intraocular pressure and cataracts		
□ osteoporosis		
☐ pituitary-adrenal suppression		
☐ psychological disturbances (eg euphoria,		
depression, paranoid psychosis)		
☐ skin atrophy		

Parenteral corticosteroids

Hydrocortisone and Dexamethasone are used intravenously for the acute treatment of asthma. The exact time course of action is not well established, but response takes at least some hours to develop.

Approximate dose equivalents of oral and parenteral corticosteroids are:

Oral prednisolone 25mg

IV hydrocortisone 100mg

IV dexamethasone 4mg

NB Prednisone is converted to prednisolone in the liver and is therefore should be used in the same dose as prednisolone

Evidence suggests that moderate- to high-dose oral corticosteroids may be as effective as parenteral corticosteroid treatment for the management of acute asthma.

Adverse effects: Very few acute adverse effects are seen, but psychoses, mood changes, hypokalaemia and hyperglycaemia can occur.

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Antihistamines

Introduction

Histamine and many other inflammatory mediator compounds are released from mast cells during type 1 (IgE-mediated) allergic reactions. Histamine released in this way stimulates H₁-receptors, which contributes to the signs and symptoms of this type of allergic reaction (eg redness, swelling, itching, sneezing, runny nose, nasal congestion, red eyes). Histamine H₁-receptor antagonists can be divided into 2 subgroups depending on their CNS effects: sedating and less sedating. **Promethazine** is available on the Tuvalu EML belongs to the first group and produces drowsiness and sedation

.Adverse effects, interactions and precautions: These drugs may affect psychomotor performance and the ability to drive motor vehicles or to operate heavy machinery. Patients must be advised of this, and cautioned against these activities. These drugs also potentiate the effect of other CNS depressants (eg alcohol).

Promethazine also has anticholinergic activity and may produce dry mouth, blurred vision, constipation and urinary retention. Its use may lead to a drying effect throughout the respiratory tract and a thickening of bronchial mucus. It should not be used where their anticholinergic activity may be contraindicated (eg in patients with narrow angle glaucoma or prostatic hypertrophy).

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Use of respiratory drugs in competitive sport

Many drugs used in the management of respiratory illnesses may be banned or restricted in the context of competitive sport. Examples include some bronchodilators, corticosteroids and decongestants.

The following drugs in the Tuvalu EML have been permitted for use in national and international sporting competition by patients with asthma:

salbutamol,
beclomethasone,
prednisolone, (when used out of competition)

In all instances, it is recommended that athletes contact their national sporting organisation before taking medication. In many cases written notification is needed via an Abbreviated Therapeutic Use Exemption form The International Olympic Committee (IOC) < http://www.olympic.org requires that athletes wishing to take asthma medications during the games have either significant bronchodilator response or a positive bronchial challenge test.

Drug-induced lung disease

Introduction

The number of drugs that have been shown to damage the respiratory system continues to grow. Drugs from the same pharmacological category tend to induce the same adverse effects. A thorough medication history should be undertaken and an adverse reaction considered in the differential diagnosis of unexplained lung disease 1].

Examples of drugs that can cause lung disease

Reaction	Drug
Bronchospasm	beta blockers, contrast media, nonsteroidal anti-inflammatory drugs
Cough	angiotensin converting enzyme inhibitors
interstitial lung disease	amiodarone, methotrexate, nitrofurantoin
Pleural effusion	amiodarone, propranolol, bromocriptine, , nitrofurantoin
systemic lupus erythematosus	hydralazine, isoniazid, , phenytoin

Inhalational drug delivery devices

Introduction

Many respiratory drugs are delivered topically to the airway by inhalational devices; this achieves an effect on the airways with a rapid onset of action and minimal systemic adverse effects.

The devices available for drug delivery are metered dose inhalers (MDIs), commonly known as 'puffers' and used with or without spacers, and nebulisers.

Errors of technique occur with all devices, so it is important to check patient technique at each review. Demonstration and repetition are essential for achieving optimal patient technique.

Metered dose inhalers (MDIs) / Puffers

Introduction

A metered dose inhaler (MDI) or puffer is a multidose device usually containing micronised powdered medication and a propellant system such as hydrofluoroalkane (HFA). Care of these devices is important:

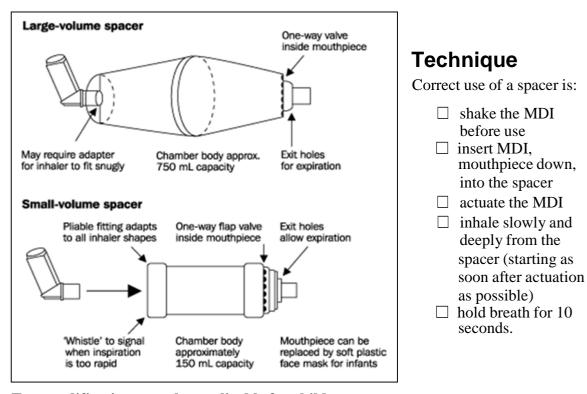
The majority of puffers need to be washed regularly to avoid blockage.
The recommended frequency of washing ranges from daily to monthly, depending on the device; refer to the specific product information for directions.
Patients should shake the device every time they use it.
If there appears to be very little liquid inside the canister when shaken, it is time to replace it
Technique
up to 70% of patients use an incorrect technique with a puffer, resulting in uate drug delivery to the lungs,
check patient technique and demonstrate the correct technique (if necessary) at every opportunity. It has been shown that there is deterioration in technique within 2 months of correct demonstration.
The device should be held upright with the mouthpiece at the bottom. This allows an accurate dose to be dispensed into the actuator valve.
Deposition of the drug from the inhaler to the airway is achieved by coordinating the actuation of the puffer and inhalation of the aerosol mist.
Starting at the end of a normal expiration , the puffer should be actuated once, at the same time as a slow deep inspiration through the mouth is undertaken. At the completion of the slow deep inspiration, the breath should be held for approximately 10 seconds.

There are two techniques which are both satisfactory if performed well:

* clo	sed mouth where the lips are sealed around the mouthpiece of the MDI
	open mouth where the inhaler is held up to 6 cm away from the open mouth.
Comm	non errors when using puffers include:
	 ☐ failing to coordinate the puffer actuation with the start of the inspiration ☐ inspiring too rapidly ☐ closing the mouth and then inspiring through the nose after actuation
	of the puffer □ actuating the puffer more than once during the inspiration □ failing to hold the breath.
Sp	acer devices
aerosolinhalat particle Spacer coordin medica	ten appropriate to use a chamber device with the MDI. These spacers hold the l cloud, which is produced from an MDI, in a confined space and allow subsequent ion over a longer period. Evaporation of some of the propellant produces es of smaller size and gives the potential for greater endobronchial deposition. devices have a valve system, which can help patients who have problems with nation. They are particularly useful in decreasing the oropharyngeal deposition of ation and increasing the proportion of the dose delivered to the lung. With inhaled esteroids, spacers are an important means of reducing candidiasis and dysphonia.
poss	alation of aerosol from the spacer should commence as soon after actuation as sible to minimise deposition in the spacer and loss of drug. One actuation of I per inhalation is recommended.
_	r devices with MDI in appropriate doses may be substituted for nebulised ation during asthma exacerbations:
	4 to 10 inhalations of standard dose short-acting beta ₂ agonists can produce a similar bronchodilator effect to standard nebulised doses.
Spacei	rs should be washed before initial use and at least monthly thereafter:
	Use warm water with kitchen detergent, Leave to drain (without rinsing) and allow to dry before use,. A cloth should not be used to dry the spacer, as this can produce an electrostatic charge causing drug particles to adhere to the walls of the spacer.

☐ Before using the spacer, it should be 'primed' by actuating 3 to 5 doses of drug; this minimises fluctuations in dose due to variation in electrostatic charge.

Spacer devices (Figure 3)



Two modifications may be applicable for children.

□ take 4 to 6 tidal breaths to inhale the aerosol
 □ use a face mask adapter to inhale from the spacer (infants and young children).

Nebulisation

Introduction

There is a tendency to overuse this expensive form of drug delivery. The inhalation, via a large-volume spacer, of 4 to 10 separate actuations from a standard beta₂ agonist MDI, provides an equivalent bronchodilator effect to that achieved by nebulisation.

Nebulisation aims to produce an aerosol from a solution of drug in a bowl This may be done using a simple pump or, if electricity is unavailable, an oxygen cylinder. In children with asthma oxygen, if available, may be the better choice.

Technique

Nebulisers produce reasonable aerosols with a flow of at least 8 L per minute. The nebuliser fill volume should be 2.5 to 5 mL of solution, which will usually achieve nebulisation of about 80% of the contents within the first 5 to 10 minutes. If nebulisation is incomplete after 10 or 15 minutes, the nebuliser might be blocked or cracked, or the pump may be faulty. Pumps should be serviced, and filters changed regularly—every 6 to 12 months depending on the amount of use.

Use of devices in children

MDIs with a spacer and mask can be used in children younger than 2 years of age. MDIs alone require a reasonable amount of coordination; therefore, they should not be used without a spacer..

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OXYGEN THERAPY

Oxygen is essential for human metabolism and lack of oxygen is generally fatal within 5 to 6 minutes. Oxygen has almost no adverse effects in the acute situation and should not be withheld if there is any suggestion of it being needed. The indications for oxygen therapy are:

- a) respiratory arrest
- b) Hypoxia of any cause
- c) acute asthma attack
- d) exacerbation of COPD

Oxygen therapy should be monitored with pulse oximetry and blood gas estimation if available. Aim to achieve an oxygen saturation of at least 95%. Humidification of oxygen is not necessary.

Methods of Oxygen Delivery

a) Intranasal Catheters

These provide a low concentration of oxygen of between 25 and 40%. They should be used with an oxygen flow rate of between 1 and 4 litres/minute (1 - 2L/min in children). Higher flow rates cause drying of the nasal mucosa and are uncomfortable. They should only be used in patients with mild hypoxia or cardiac failure or myocardial ischaemia. They do not provide a high enough oxygen concentration for patients with significant hypoxia, carbon monoxide poisoning, shock or cardiac arrest.

b) Plastic Face Masks

These provide oxygen concentrations of between 35 and 70%. The oxygen flow rate should be set between 4 and 15 litres/minute. Do not use face masks with an oxygen flow rate less than 4 litres/minute. This method of oxygen delivery is suitable for patients with moderate hypoxia or shock.

c) Tight Fitting Face Masks (eg. Laerdal, CPAP masks)

These devices can provide oxygen concentrations close to 100%. They should be used in patients with severe hypoxia or with cardiac arrest.

Adverse Effects of Oxygen

Patients with chronic obstructive airway disease and elevated carbon dioxide levels may occasionally have a hypoxia-dependent respiratory drive. In these patients, the administration of oxygen causes hypoventilation and an increase in the carbon dioxide level. Although this may cause problems it is far less dangerous than hypoxia itself. In the emergency situation, it is important that hypoxia is corrected - problems with carbon dioxide retention can be handled later. Do not hesitate to give oxygen to hypoxic patients with chronic obstructive airway disease.

Administration of 100% oxygen sometimes causes pulmonary toxicity but this only occurs after 24 hours and therefore is not a problem in the emergency situation.

NOTE: If arterial blood gases are available, then they should be measured before the commencement of oxygen to establish the baseline.

Pulmonary function testing

Introduction
Pulmonary function tests play a role in:
 □ assessing breathlessness, asthma and other chronic chest disorders □ monitoring response to treatment □ assessing fitness for surgery
The results are reported in relation to reference values and whether they fall within the normal range for an individual of that age and gender
Available tests measure expiratory air flow.
Peak evniratory flow (PFF)

Peak expiratory flow (PEF)

Uses a portable PEF meter and can be valuable in assessing the diurnal variability of airflow obstruction (a characteristic feature of asthma), as well as the response to therapy.. The technique is simple and can be performed as part of the asthma management plan. It is not as sensitive as spirometry but patients can be taught to do it.

Technique is important:

insert tube firmly ino the monitor
hold a few centimetres from the mouth
take inand hold as deep a breath as possible
put tube into mouth and close lips firmly around it
blow into tube as hard, fast and long as possible
Rest for 30 seconds
Repeat above steps twice
Record best result

Spirometry

This measures more parameters of lung function and is mor accurate than PEF. However the test is effort-dependent, and should be performed by trained personnel in order to obtain reproducible results.

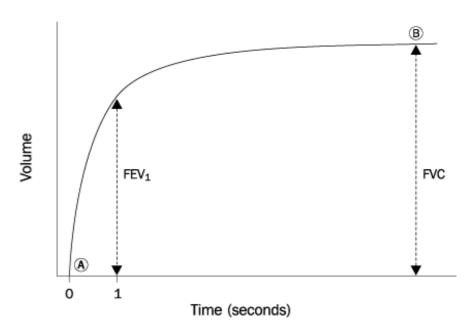
It should be performed using the same technique as for PEF

For immediate assessment and ongoing monitoring of asthma the forced expiratory volume exhaled in 1 second (FEV₁) is the most useful. For assessment of lung damage in a variety of lung diseases, the expiratory forced vital capacity (FVC) and the total lung

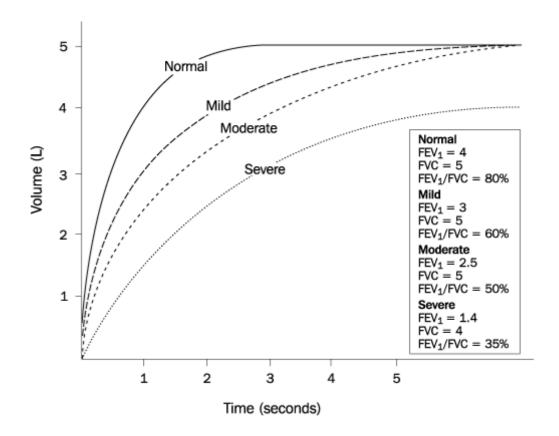
capacity (TLC) can be helpful. Additionally the FEV_1 can be recorded and expressed as a percentage of FVC.

Results for an individual can be compared with reference values matched for age, gender, height and ethnicity. The range of normal values for the FEV₁ and FVC is between 80% and 120% of that predicted from the reference values.

Spirogram



Spirogram showing obstructive ventilatory defect



Pulse oximetry

Pulse oximetry measures oxyhaemaglobin saturation (SaO ₂) by applying a clip containing
light-emitting diodes to the finger or earlob. The method is non-invasive and, where
available, can provide very helpful information in the diagnosis of severity of respiratory
disease. It can also be helpful in assessing progress in the management of acute asthma and respiratory failure.

Asthma

Introduction

Asthma is a common chronic inflammatory disorder of the airways in which many cells and cellular elements play a role. The chronic inflammation causes an associated increase in airway hyperresponsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread but variable airflow obstruction that is reversible either spontaneously or with treatment'

Management includes prevention of attacks, treatment of acute attacks and, where necessary, long term medication which works best with strict adherence. All patients, both adult and children, should be given an asthma plan which needs frequent review and repetition to ensure full understaning by the patient or parents.

It is essential that they understand the difference between the blue reliever puffers (MDIs) and the brown preventer puffers.,

Prevention of attacks

It may be possible to prevent or reduce the severity of attacks by:

☐ avoidance of trigger factors where possible eg known allergens, tobacco smoke, stress etc
☐ appropriate management of acute exacerbations
☐ appropriate management of infections
appropriate long-term medication use with an individual patient
management plan
☐ Regular assessment by lung function tests

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☐ Avoidance of drugs that can cause bronchoconstriction eg beta blockers, aspirin, ibuprofen and indomethacin.

NB Paracetamol is rarely a problem and is the analgesic of choice for asthmatics.

Treatment of an acute attack of asthma

This must always be treated as a serious emergency

Severity is estimated by clinical assessment, measurement of peak expiratory flow rate and by pulse oximetry.

Pulse rate may also be helpful in indicating severity of an attack:

☐ Mild <100

☐ Moderate 100-120 (children 100-200)

☐ Severe >120 (children >200)

Wheezing is an unreliable indicator of the severity of an asthma attack and may be absent in a severe attack. In severe asthma the patients will lack sufficient air flow to perform lung function tests

Cyanosis indicates life-threatening asthma.

All patients with moderate or severe asthma should be given oxygen. Patients with severe asthma should be managed in an intensive care unit if possible and may occasionally require intubation and mechanical ventilation.

In ADULTS:

Oxygen

Give oxygen via face mask if moderate or severe asthma.

Beta-adrenergic Agonists

Use beta-adrenergic agonists to reverse bronchospasm. In very severe asthma intravenous salbutamol may be useful in addition to nebulized.

Give salbutamol 5 mg by nebulizer with oxygen and repeat every 30 minutes if necessary (or give continuously in severe asthma)

OR

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Give salbutamol by puffer using spacer (up to 50 puffs) if nebulisers are not available

PLUS if very severe

Give salbutamol 5 microgram/kg intravenously (to a maximum of 250 microgram) over one minute then commence an infusion at 5 microgram/kg per hour

NOTE: Continuous nebulized salbutamol is probably as effective as intravenous

Anticholinergics

These agents have a synergistic effect with beta-adrenergic agonists. In severe asthma consider the use of ipratropium.

Give ipratropium bromide 0.5 mg by nebulizer and repeat every 4 hours if necessary

Corticosteroids

Steroids reduce inflammation of the airways. Their effects are delayed for at least 4 hours but they are important to prevent relapse. Oral steroids are as effective as those given parenterally in most patients.

Give hydrocortisone 200 mg intravenously then 100mg six hourly

OR

Give prednisolone 50 mg orally daily

Other Drugs

Aminophylline provides no additional benefit to optimal doses of beta-adrenergic agonists. It has a number of undesirable side effects including seizures, ventricular tachycardia, hypokalaemia and vomiting. Routine use in asthma is not recommended. However, it may be of benefit in patients with severe asthma who require hospitalisaton. A loading dose is given to patients who are not taking oral theophylline:

Give aminophylline 5 mg/kg (to a maximum of 250 mg) intravenously over 5 minutes

Adrenaline does not appear to have any advantage over salbutamol. It may be used as a last resort or when intravenous access or nebulisers are not available:

Give 1:1000 adrenaline 0.5 - 1 mL intramuscularly or subcutaneously

NOTE: Adrenaline may be given down the endotracheal tube - the dose is 5 times the intravenous dose and it should be diluted in 10 ml of normal saline.

In CHILDREN:

Oxygen

Give oxygen via face mask to all children with asthma.

Beta-adrenergic Agonists

Use beta-adrenergic agonists to reverse bronchospasm. In very severe asthma intravenous salbutamol may be useful in addition to nebulized.

Give salbutamol 2.5 mg by nebulizer with oxygen to children 5 years of age or under, or give 5 mg by nebulizer to children over 5 years and repeat every 30 minutes if necessary (or give continuously in severe asthma)

PLUS if very severe

one minute then commence an infusion at 5 microgram/kg per hour

NOTE: Intravenous salbutamol may be more effective than continuous nebulized in young children with severe asthma.

Anticholinergics

These agents have a synergistic effect with beta-adrenergic agonists.

Give ipratropium bromide 0.25 mg by nebulizer and repeat every 4 hours if necessary

Corticosteroids

Steroids reduce inflammation of the airways. Their effects are delayed for at least 4 hours but they are important to prevent relapse. Oral steroids are as effective as those given parenterally in most patients.

Give hydrocortisone 1 - 4 mg/kg intravenously to a maximum of 200 mg then every six hourly

OR

Give dexamethasone 0.2 mg/kg intravenously or intramuscular to a maximum of 8 mg

OR

Give prednisolone (prednisone) 1 mg/kg orally to a maximum of 50 mg daily

Other Drugs

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Give aminophylline 5 mg/kg (to a maximum of 250 mg) intravenously over 5 minutes

Adrenaline does not appear to have any advantage over salbutamol. It may be used in severe asthma as a last resort or when intravenous access is not available:

Give 1:1000 adrenaline 0.1 ml/kg intramuscularly or subcutaneously to a maximum of 0.5 ml

Management after an acute attack

Adul	lts —
All patients v	vill need follow up and some form of on going therapy
	 □ review trigger factors to identify the possible cause of the attack □ discuss avoidance measures □ start or adjust the patient's maintenance therapy □ design or adjust the patient's asthma action plan □ review adherence to prescribed medication regimen.
Continu	uing Management
Mild Epi	sodic
\square salb	may only need to use: outamol puffer 1-2 puffs up to 8 puffs a day. (with spacer for children or lts with poor coordination)
med	uld all be advised of the symptoms of an acute attack and told to seek dical advice if their need to use the puffer increases or they are using more a 8 puffs a day
OR	
if unable GIVE	to use a puffer with or without a spacer
	albutamol tablets 4mg 6-8hourly

OR	
	Theophylline SR 300mg orally bd
Mod	lerate Persistent
	salbutamol puffer 1-2 puffs prn up to 8 puffs a day. (with spacer for children or adults with poor coordination)
OR	
if un	able to use a puffer with or without a spacer GIVE
	Salbutamol tablets 4mg 6-8hourly
OR	
	Theophylline SR 300mg orally bd
ANI	O ADD
	beclomethasone diproprionate 100 microgram bd
Reviev	N .
	 □ any new trigger factors □ asthma management plan □ arrange for follow up if possible
Severe	Persistent
If	symptoms persist or deteriorate
	continue any existing oral therapy and inhaled salbutamol
	Using a spacer with 5-10 puffs inhaled over 5-10 minutes every 2 hours If available give nebulised salbutamol 5mg over 10 minutes 2-4 hourly

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☐ Increase beclomethasone diproprionate of	lose to 200 microgram bd
IF SYMPTOMS PERSIST START	
☐ Prednisolone 25-50mg orally daily for 1-	-2 weeks
If response is good tail off the predisolone a	ver 10days
Some patients with severe asthma will nee	ed:
☐ Prenisolone 5-7.5mg orally long term	
If none of the above treatments produce impr referred urgently to hospital for acute manag	<u>-</u>
Children	
All should have follow up and some form of on	going therapy and asthma plan
☐ review trigger factors to identiful discuss avoidance measures start or adjust the patient's maiul design or adjust the patient's as review adherence to prescribed	ntenance therapy othma action plan
Mild Episodic	
GIVE □ salbutamol puffer 1-2 puffs prn	up to 8 puffs a day (with spacer.)
Parents should be advised of the symptoms of medical advice if their need to use the than 8 puffs a day	
Moderate Persistent	
□ salbutamol puffer 1-2 puffs prn	up to 8 puffs a day. (with spacer)
OR	

if unable to use a puffer with or without a spacer

ANI	O ADD
For	children over 6 and/or able to use an inhaler with a spacer
	beclomethasone diproprionate 50-100 microgram bd
	For children uder 6 and/or unable to use an inhaler with spacer
	GIVE Prednisolone 1mg/Kg a day for 3 days and review
	If unable to reduce or cease prednisolone refer to a doctor
Sev	ere Persistent
If sy	mptoms persist or deteriorate
	continue any existing oral therapy and inhaled salbutamol
ANI	5-10 puffs salbutamol inhaled using a spacer over 5-10 minutes every 2 hours If available give nebulised salbutamol 5mg over 10 minutes 2-4 hourly Increase beclomethasone diproprionate dose to 100 microgram bd
OR	for children unable to inhale with a spacer
GIV	E
	Prednisolone 2mg/kg orally daily for 1-2 days

If none of the above treatments produce improvement the patient should be referred urgently to hospital

Review and education

Introduction

Optimal self-management and education with regular clinical review results in significant reductions in emergency health care utilisation. A structured program, conducted over time, that teaches detection and management of deteriorating asthma and optimal use of medications is required. Essential components are:

	☐ written information about asthma
	☐ self-monitoring and feedback
	☐ education about optimal delivery device technique (see <[Inhalational drug delivery devices]).
	provision of an individualised written asthma action plan (see <[Asthma action plan])
	☐ regular medical review and assessment of medications (including corticosteroid dose reduction when the patient has been stable for a reasonable length of time, eg 3 months)>.
Adheren	ce (compliance)
	ol is poor despite apparently adequate treatment, consider poor adherence. s with improvement in symptoms. Strategies that may improve adherence
	 □ utilising an open, nonjudgmental approach when discussing adherence □ allowing the patient to express their concerns about medication, and addressing these concerns □ improving the patient's understanding of asthma management over a period of time. Comprehensive information can rarely be retained after one visit □ explaining the goals of treatment. Adherence will be improved if your aims are in concordance with the patient's goals □ keeping treatment simple and setting achievable goals in collaboration with the patient (using once- or twice-daily dosing where possible, using as few medications as possible) □ discussing potential adverse effects. For further information, see □ Getting to know your drugs □ identifying useful daily associations to simplify medication adherence (eg using preventive therapy in the bathroom, and then brushing teeth)
	 □ obtaining support of the patient's family and peers □ keeping in touch.
	 □ need for infants or toddlers to use oral or inhaled corticosteroids □ the need for older children to use maintenance inhaled corticosteroid therapy at doses greater than 600 micrograms daily of beclomethasone (or the equivalent dose of other drugs) □ the need for a discussion on complications of treatment □ suspected occupational asthma □ the need for a detailed discussion on control of the home environment
	☐ the need for a detailed discussion on control of the home environment.

Asthma management plan In the management of asthma, both patients and health professionals should use the same framework of management and similar terminology. To facilitate this, a 6-point management plan⁶ has been proposed: ☐ Assess asthma severity, ☐ Achieve best lung function ☐ Maintain best lung function: identify and avoid trigger factors ☐ Maintain best lung function: optimise medication program ☐ Develop an asthma action plan Educate and review regularly Asthma action plan All patients should have an asthma action plan—in written form—that outlines how to: ☐ recognise symptoms of asthma deterioration ☐ start treatment ☐ reach medical attention. Action plans may be based on peak expiratory flow (PEF) measurements or asthma symptoms or both. Plans should be simple, individualised and based on 2 to 4 action points. Plans based on PEF measurements should use personal best PEF as for action points. All patients with asthma should know how to obtain prompt medical assistance. The principles of asthma action plans are: increase the dose and frequency of inhaled beta₂ agonist ☐ increase the dose of inhaled corticosteroid or commence prednisolone if the patient is already on a high dose of inhaled corticosteroid ☐ obtain prompt medical attention ☐ in an emergency, immediate use of a high-dose inhaled short-acting beta2 agonist (eg salbutamol 6 to 10 inhalations by MDI or 5 mg by nebuliser) and transfer to an emergency department (preferably by an ambulance that carries supplemental oxygen).

Asthma action plans must be individualised.

⁶ By the Thoracic Society of Australia and New Zealand, The Royal Australian College of General Practitioners, the Pharmaceutical Society of Australia and the Asthma Foundations, through the National Asthma Council.

Chronic obstructive pulmonary disease (COPD)

Definitions

Chronic obstructive airways disease (COPD) is characterised by airflow obstruction that is not fully reversible. The airflow limitation is in most cases both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases, most commonly cigarette smoke. COPD is usually some combination of:

□ em	physema, where the lung parenchyma is structurally damaged, with
dest	ruction of alveolar septa and formation of abnormally enlarged
airsį	paces
□ airv	way damage with airway wall thickening and narrowing of the
airw	ay.
	ronic bronchitis which is defined clinically as a cough productive of um, occurring on a daily basis for 3 months in each of 2 consecutive
year	·S

Some patients may have bronchodilator responsiveness whether or not they have a history of asthma. The dyspnoea of COPD is frequently associated with cough, sputum production, recurrent respiratory infection and wheezing, which may only be evident during infective exacerbations. Typically, the dyspnoea has developed insidiously over several years and it may be the patient's only symptom.

Typically, COPD affects middle-aged and older people, and **cigarette smoking is the major causative factor.** The figure illustrates the accelerated decline in lung function caused by smoking.

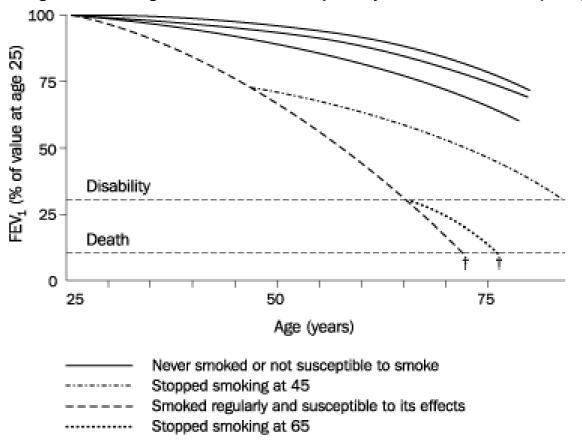


Figure 4.Smoking and loss of forced expiratory volume in 1 second (FEV₁)

The differences between the lines illustrate effects that smoking, and stopping smoking, can have on the FEV_1 . This figure shows the rate of loss of FEV_1 for one particular susceptible smoker; other susceptible smokers will have different rates of loss, thus reaching 'disability' at different ages.

† = death, the underlying cause of which is irreversible chronic obstructive pulmonary disease, whether the immediate cause of death is respiratory failure, pneumonia, cor pulmonale or aggravation of other heart disease by respiratory insufficiency

This figure was first published in Fletcher C, Peto R. The natural history of chronic airflow obstruction. BMJ 1977;1:1645-8 and is reproduced by permission of the BMJ Publishing Group.

Solid line mean and SD Decide locally how much information to include but will need reference

Measurement of lung function

The spirometric abnormalities associated with COPD are a reduction in postbronchodilator forced expiratory volume in 1 second (FEV₁), and a reduction in the FEV₁/forced vital capacity (FVC) ratio to less than 70% (ie an obstructive pattern).

Management of chronic stable COPD

Smoking cessation

Smoking cessation is the only intervention that has been shown to improve the natural history of COPD; to prevent deterioration it is vital that the patient stops smoking. (see figure 3)

Bronchodilators for long-term treatment of COPD

Introduction

In the long-term treatment of COPD, bronchodilators are recommended for the relief of wheezing and shortness of breath. Anticholinergic agents are more effective in COPD than they are in asthma.

Bronchodilators can improve the FEV₁, FVC and exercise tolerance independently of each other. Spirometric changes are not seen in all patients, but improvement in symptoms and functional capacity can occur even without spirometric changes.

GIVE
\square salbutamol 100 to 200 micrograms MDI by inhalation, up to 4 times daily
OR
\Box ipratropium bromide 40 to 80 micrograms by inhalation, up to 4 times daily.
If the response to initial single-agent therapy is unsatisfactory or the patient has moderate to severe disease,
GIVE
$\ \square$ salbutamol 100 to 200 micrograms MDI by inhalation, up to 4 times daily
PLUS
\Box ipratropium bromide 40 to 80 micrograms by inhalation, up to 4 times daily.
Patients with poor inhalation technique can use a large-volume spacer; this improves lung deposition of the aerosol
In patients who are unable to use an MDI (with or without a spacer),
USE
☐ Salbutamol 4mg orally up to 8 hourly as tolerated
OR
☐ Theophylline SR 300mg bd

The effect of these agents may be monitored by self reported symptoms or by PEF or spirometry.

Corticosteroids for long-term treatment of COPD Introduction

Only 10% of patients with stable COPD benefit in the short term from corticosteroids. There are no distinguishing clinical features to predict which patients may benefit. Oral corticosteroid reversibility tests do not predict response to inhaled corticosteroids; they should not be used to identify which patients should be prescribed inhaled corticosteroids.

Inhaled corticosteroids

The aim of treatment with inhaled corticosteroids is to reduce exacerbation rates and slow the decline in health status, not to improve lung function *per se*. The effect of inhaled corticosteroids on mortality is uncertain.

Benefits are not seen in patients with COPD who continue to smoke cigarettes.

inhaled cortic	osteroids should be prescribed for patients:
	\square with an FEV ₁ less than or equal to 50% predicted
	☐ who have documented evidence of responsiveness to inhaled corticosteroids (on PEF or spirometry)
	□ who have had 2 or more exacerbations requiring treatment with antibiotics or oral corticosteroids in a 12-month period.
GIVE	
	☐ beclomethasone dipropionate 300 to 400 micrograms by inhalation, twice daily

To minimise oropharyngeal candidiasis and systemic absorption of inhaled corticosteroids, patients should be advised to rinse their throat and mouth with water and spit out after inhalation. Patients using a puffer should also be advised to use a spacer to lessen the risk of candidiasis and dysphonia.

The response to corticosteroids should be closely monitored with measurement of PEF or spirometry.

Patients who do not suffer frequent exacerbations should be assessed after about 6 weeks of treatment. Because of the potential risks of long-term corticosteroid use, only those patients in whom clear objective benefit has been obtained should continue with treatment. Inhaled corticosteroid doses should be gradually reduced to the minimum dose that maintains subjective benefit.

Oral corticosteroids

Oral corticosteroids are not recommended for maintenance therapy in COPD. However, they may be needed in some patients with advanced COPD in whom corticosteroids cannot be withdrawn following an acute exacerbation. In these cases, the dose of oral corticosteroid should be kept as low as possible.

Combined therapy for long-term treatment of COPD

If patients remain symptomatic on monotherapy, their treatment should be intensified by combining therapies from different drug classes. Combined therapy should be discontinued if there is no benefit in symptoms or lung function tests..

Management of pulmonary hypertension and cor pulmonale

Cor pulmonale is defined as an alteration in the structure and function of the right ventricle co W f Ca co

common link be with COPD de causing symptom	between lung dysfunction and the heart in cor pulmonale. Hypoxic patients velop pulmonary hypertension, which may be present for years without oms. In some patients it leads to the development of the clinical syndrome of This should be considered if patients with COPD have:
	 □ peripheral oedema □ raised jugular venous pressure □ a systolic parasternal heave □ a loud pulmonary second heart sound.
GIVE	
	☐ Frusemide 40-80mg orally daily
If this is insu	fficient to control symptoms and signs
ADD	
	☐ Enalapril 2.5mg daily followed by gradual increments to a maximum of 20mg daily
dehydrate	can result after the first dose in the elderly, patients who are already d by diuretic therapy, in the presence of pre-existing hyponatraemia and t treatment with other anti-hypertensive drugs.
Obstruc	ctive sleep apnoea
advanced disea	eep apnoea is common in patients with COPD, particularly in those with ase. Patients with COPD and coexisting sleep apnoea may have hypoxaemia uring sleep and in the recumbent position (see <[Obstructive sleep apnoea]).
tailored and de autonomy.	signed to optimise the patient's physical and social performance and

Self-management plan

Patients at risk of having an exacerbation of COPD should have a self-management plan and be encouraged to respond promptly to the symptoms of an exacerbation. This plan should outline the initial response the patient should take, and may include:

if breathlessness increases-	-adjust bronchodilator	therapy to contro
symptoms		

 ☐ if breathlessness increases and interferes with activities of daily living—start oral corticosteroid therapy (unless contraindicated) ☐ if sputum increases in volume or becomes purulent—start antibiotics.
These patients should keep a course of antibiotic and corticosteroid tablets at home for use
Antitussive therapy
Sipping hot water with or without honey, lemon and ginger can be soothing but other antitussive therapy should not be used in the management of stable COPD. Antitussive therapy may suppress breathing and induce hypercapnia, and can be counterproductive by causing sputum retention and constipation.
Prophylactic antibiotic therapy
There is insufficient evidence to recommend prophylactic antibiotic therapy in the management of stable COPD.
Acute Exacerbation of Chronic Obstructive Pulmonary Disease
Exacerbation of chronic obstructive airways disease (COPD) is a common problem in emergency medicine. The response of COPD to treatment is generally slower than that of asthma and most patients require admission.
Oxygen
It is essential that oxygen be given to maintain oxygen saturation greater than 92%. Although administration of oxygen can cause an elevation in arterial carbon dioxide levels in a few patients, this is far less of a problem than hypoxia itself. For mildly hypoxic patients oxygen via an intranasal catheter will be sufficient while those with more severe hypoxia may require oxygen via a face mask. Use the lowest flow rate necessary to maintain an adequate arterial oxygen saturation.
CAUTION: In patients with C02 retention, oxygen saturation should be maintained between $90-95\%$.
Bronchodilators
Salbutamol and ipratropium have a synergistic action:
☐ Give salbutamol 5 mg via nebulizer every 2 to 4 hours

If a nebuliser is not available use puffers with a spacer

 \square Give ipratropium bromide 0.5 mg via nebulizer every 4 hours

PLUS

Corticosteroids

Oral and parenteral routes are equally effective except in the sickest patients.
☐ Give hydrocortisone 200 mg intravenously every 6 hours
OR
☐ Give prednisolone 40 mg orally daily
Antibiotics
Since many exacerbations of chronic obstructive pulmonary disease (COPD) are due to viral infection or noninfective causes, antibiotics are only occasionally indicated. At least half of patients with chronic bronchitis are persistently colonised with <i>Haemophilus influenzae</i> , <i>Streptococcus pneumoniae</i> or <i>Moraxella catarrhalis</i> —hence
a positive sputum culture is not necessarily indicative of acute infection.
However, these organisms may be responsible for more severe exacerbations, in which antibiotics have been shown to be of benefit. The aim of treatment with antibiotics in acute exacerbations of chronic bronchitis is to reduce the volume and purulence of sputum; elimination of colonising organisms is not required.
Antibiotics have been shown to be effective only when all 3 cardinal symptoms of acute bacterial exacerbations are present: increased dyspnoea, increased sputum volume and sputum purulence.
When indicated, use
☐ .Give amoxycillin 500 mg orally or ampicillin 500mg intravenously every 8 hours
OR if penicillin sensitive
☐ Give erythromycin 500 mg orally every 6 hours for 5-7 days
Cough

Introduction

Cough is a frequent symptom and sign of an underlying disease, but is not itself a diagnosis. Management should concentrate on defining and then treating the underlying cause, if appropriate and possible.

Cougn may be due to:
☐ Smoking
☐ Infections eg viral sore throat,tracheitis, pneumonia,tb etc
☐ Asthma
☐ Rhinitis/postnasal drip
☐ Oesophageal reflux
☐ Aspiration eg neuromuscular disorders, stroke
☐ Drugs eg angiotensin converting enzyme inhibitors, beta blockers
☐ Carcinoma of bronchus or lung
☐ Bronchiectasis
☐ Interstitial lung disease
☐ Heart Failure, especially if cough nocturnal
Cough in Adults
The most important aspect of management is the diagnosis treatment of any underlying condition and reassurance where appropriate.
Cough Linctuses and cough suppressants are of little value and sipping hot water, with or without lemon, honey and ginger is as effective as anything.
If cough is associated with a sore throat or upper respiratory infection GIVE
☐ Paracetamol 1g orally 6 hourly – maximum dose 8g a day for no more than 3 days

Paediatric aspects of cough

Causes

Introduction

Many of the common causes of cough in adults occur in children. More so than in adults with chronic cough, there is considerable controversy regarding the importance of postnasal drip and gastro-oesophageal reflux in the aetiology of chronic cough in children. Some additional factors that need to be considered are outlined below.

Infections

Recurrent viral bronchitis is the most common cause of recurrent cough in children.

Specific infections, such as pertussis, TB and those caused by *Mycoplasma pneumoniae*, cause cough as a part of typical clinical syndromes. *Chlamydophila* (formerly known as *Chlamydia*) infection may cause a prolonged cough in the first few months of life.

CROUP

Croup is a viral infection of the upper airway which affects children from the ages of 6 months to 3 years. It is characterised by fever, a harsh cough, a hoarse voice and stridor. Children who have stridor while at rest or who have signs of respiratory distress (i.e. suprasternal retraction, tachypnea, restlessness) should be admitted. Pulse oximetry is useful – an oxygen saturation of 93% or less while breathing air is also an indication for admission. Most cases of croup however are mild and self-limited.

Mild croup

These patients will have stridor only with exertion or crying and no signs of respiratory
distress. Avoid exposure to cold air. Give paracetamol for fever.
☐ Give paracetamol 20 mg/kg every 4 hours
Moderate croup
These patients will have stridor at rest and some signs of respiratory distress but oxygen saturation should be greater than 90% on air.
\square Give oxygen to maintain an oxygen saturation greater than 93%
PLUS
☐ Give dexamethasone 0.6 mg/kg intramuscularly as a single dose

Severe croup

These patients will have signs of marked respiratory distress plus hypoxia or cyanosis. Admission to an intensive care unit is desirable and intubation may be necessary.	
	Give oxygen to maintain an oxygen saturation greater than 93%
	PLUS
	Give dexamethasone 0.6mg/kg intramuscularly as a single dose(SUBSTITUTE WITH HYDROCORTISONE)
	PLUS
	Give nebulized adrenaline, 0.5 ml/kg of 1: 1000 solution or 0.05 ml/kg of a 1% solution diluted with saline to a volume of 2.5 ml

NOTE: Patients who fail to respond to nebulized adrenaline may require endotracheal intubation. Nebulized adrenaline provides only temporary relief of airway obstruction lasting 1 to 2 hours. Patients should be closely observed after this period for recurrence of obstruction.

'Asthma'

In the absence of wheeze or evidence of reversible airway obstruction, the presence of cough alone is unlikely to be due to asthma. Recent studies suggest that many children with these symptoms have increased cough receptor sensitivity secondary to viral respiratory infections. These children do not generally respond to antiasthma medications, especially inhaled corticosteroids.

Mechanical causes

A persistent unproductive irritant cough with no obvious cause may be an indication for bronchoscopy to exclude the following: a foreign body, an unusual focal lesion of the bronchial wall, or an extrinsic lesion pressing on the airway.

Aspiration of milk should be considered as a cause in infants when cough is related to feeding.

Smoking

Smoking should be considered as a cause of cough in children: 10% of children are regular smokers by the age of 12 years. Environmental tobacco smoke exposure from parental smoking may also be a cause of cough, especially in children under 2 years of age.

].

	medications have a very limited place in paediatric practice. sk for something to soothe the child's throat
	☐ Hot water with honey, lemon and ginger. This may be sipped as often as required
Upper Re	spiratory Tract Infections
Rhinitis	
complaining of drip, itching of (eg chronic sin	mon, affecting approximately 20% of the population. In a patient rhinitis symptoms—sneezing, rhinorrhoea, nasal obstruction, post-nasal the nose—it is important to exclude any underlying or associated pathology usitis, nasal polyps,) before commencing drug treatment. Unilateral foularge, especially in children, may indicate a foreign body in the nasal cavity.
Causes of rhini	tis include:
	 □ infections—viral, bacterial or fungal □ environmental allergens □ occupational exposures (eg wood dust, grains, chemical, latex, aerosols of nickel salts) □ drug use (eg aspirin, NSAIDs, antihypertensives, oral contraceptives), drug abuse (eg cocaine sniffing), □ hormonal changes related to menstrual cycle, pregnancy or puberty
	casionally be caused by emotional changes, by some foods, or by other as gastro-oesophageal reflux, especially in children.
\square In the l	iral rhinitis: common cold arge majority of cases antibiotics should not be given and management be non pharmacological
☐ Fever w	with significant facial pain and frank pus from the nose suggests sinusitis, ibiotic treatment may be indicated.
GIVE	
•	cillin 500mg orally 8 hourly for 7 days te hydration, especially in children, is the most important aspect of ement.

If fever and/or	headache are present
GIVE ADULT	ГЅ
	☐ Paracetamol 1g 4-6 hourly orally in adults. Maximum dose 8g a day for no more than 3 days for adults
GIVE CHIL	DREN
	☐ Paracetamol 20mg/kg every 4 hours for a maximum of 48 hours

Allergic rhinitis

Introduction

Over the last decade, the prevalence of allergic rhinitis has increased worldwide. It is caused by environmental allergens and can be aggravated by chemical irritants (eg active or passive smoking). If possible, establish the cause of the allergy and advise the patient on techniques to minimise exposure to both allergens and irritants. Allergic rhinitis and asthma often coexist and this association should be looked for.

Allergic rhinitis is classified as either intermittent (lasting for less than 4 days of the week **or** less than 4 weeks) or persistent (lasting for more than 4 days of the week **and** more than 4 weeks). The severity of symptoms is classified as either mild or moderate/severe.

Mild disease

Mild disease minimally impairs daily activities (including work, sport, school and leisure) and does not usually cause sleep disruption. If the patient has persistent disease with significant sleep disturbance

GΓ	IVE	
П	Promethazine 10mg	at bedtime

. Patients should be advised that sedating antihistamines may interact with alcohol and may affect ability to drive and operate machinery .

Moderate/severe disease or inadequately controlled mild disease

Rhinitis is defined as moderate/severe disease if it interferes with sleep and/or impairs activities of daily living, leisure, work, school or sport. Intranasal corticosteroids can be helpful in this situation. They have a slow onset of action and need to be used continually for maximum effect. They are effective for the relief of all rhinitis symptoms (including nasal congestion) and often relieve eye symptoms. Patients need to be advised to shake the device, and to clear the nasal passages before using the nasal spray (the use of a saline nasal spray may be helpful)

GIVE

beclomethasone dipropionate 50 micrograms/spray, 2 to 4 sprays into
each nostril, twice daily (child 3 to 12 years: 1 spray into each nostril
twice daily). The drug not available in current EML.

NB referral to an ENT clinic will be necessary to obtain necessary advice

.

Nonallergic rhinitis

The mainstay of management of nonallergic rhinitis is to try to identify any cause and correct this if possible. Intranasal corticosteroids often provide symptom relief, but may have to be continued for prolonged periods. In vasomotor rhinitis

Intractable rhinitis

If symptoms of rhinitis are continuous and not controlled by maximum doses of intranasal corticosteroids, the diagnosis should be reviewed—consider enlarged adenoids in children or chronic sinusitis in adults. Referral to an ear nose and throat surgeon may be helpful, especially if nasal polyposis that has not responded to intranasal corticosteroids is present; surgical resection may be needed.

In children who have rhinitis for more than 3 months, a bacterial infection may be present. For recommendations about choice of antibiotic and length of treatment, see the section on sinusitis in For severe and persistent allergic rhinitis in adults that has not responded to topical corticosteroids, consider

prednisolone 10 to 25 mg orally, once daily for 7 to 10 days.

However, prednisolone should not be used long-term for rhinitis. A single intramuscular injection of a depot preparation of methylprednisolone is sometimes used.

Acute Epiglottitis

Epiglottitis is a medical emergency and failure to provide prompt treatment may be fatal. It is due to infection of the epiglottis with Haemophilus influenzae bacteria. Epiglottitis mainly affects children between the ages of 3 and 8 years but is occasionally seen in adults as well. It is characterised by fever, inspiratory and expiratory upper airway noises, a severe sore throat, dysphagia and drooling. The patient usually looks very unwell.

There is a very high risk of acute airway obstruction. All patients should be referred immediately to an anaesthetist and admitted to an intensive care unit. Attempting to view the throat or otherwise upsetting the child may cause airway obstruction and should be avoided. Keep the patient sitting up.

Give □ ceftriaxone 100 mg/kg stat then 50mg/kg intravenously daily
OR

Give

\square chloramphenicol 40 mg/kg stat then 25mg/kg intravenously daily
Early transfer to oral therapy is desirable
☐ Chloramphenicol 750mg-1g IV 6 hourly for 5-10 days.
Alternative: ☐ Ceftriaxone 2g IV as a single dose daily 5-10 days (impatient use only) OR ☐ Ampicillin 500mg-1g IV 6 hourly for 5-10 days (when sensitivity known)
Lower Respiratory Tract Infections
ACUTE BRONCHITIS
In an immunocompetent adult or child, acute bronchitis is most often viral and does not require antibiotic therapy. Randomized controlled trails show that antibiotic therapy provides on overall benefit to the patient and may cause harm.
If severe and practically if sputum is voluminous and purulent, with fever, secondary bacterial infections is assumed: Amoxycillin 500mg orally 8 hours for 5-7 days.
Alternative: ☐ Doxcycline 100mg orally 12 hours for 7 days (if available) OR ☐ Tetracycline 500mg orally 6 hours for 5-7 days OR ☐ Erythromycin 500mg orally 6 hours for 5-7 days

ACUTE EXACERBATION OF CHRONIC BRONCHITIS

Acute exacerbation could be either viral or bacterial infection. Common organisms include *Strep pneumoniae*. *H influenzae*, *Moraxella catarrhalis*. The indication for Antibiotic therapy is increased cough and dyspnoea TOGETHER with increased sputum volume and/or Purulence.

Treatment as for Acute Bronchitis above.

PNEUMONIA

COMMUNITY ACQUIRED

Choice of antibiotic is often empirical. In immunocompetent, otherwise healthy patients it is usually caused by single microorganisms such as *Streptococcus pneumoniae*. *H influenzae*, *Mycoplasma pneumoniae*, *Chlamydia*. *Staph aureus* is not uncommon in Tuvalu

Note: In immunocompromised patients such as diabetics or in elderly patient or patients with co-existent illness (e.g. cancer, liver disease, heart failure or renal failure) a broad-spectrum antibiotic cover may be required.

(a)	Mild Disease
	Amoxycillin 500mg orally 8 hours for 7-10 days OR
	Procaine Penicillin 3.6g (4 mega units) IM daily for 7-10 days.
Alte	ernatives:
	Tetracycline 500mg orally 6 hourly for 7-10 days OR
	Doxycycline 100mg orally 12 hours for 7-10 days
If hy	ypersensitive to penicillin or Mycoplasma or Chlamydia suspected:
[☐ Erythromycin 500mg orally 6 hourly for 10-14 days OR
	☐ Doxycyline 100mg orally 12 hours for 10-14 days
(b)	Moderate Disease
[☐ Penicillin G 1200mg (2 mega unit) IV 6 hours for 7-10 days
In pa	atients hypersensitive to penicillin
-	☐ Chloramphenicol 1g IV 6 hourly X 7 – 10 days
subs	the clinical response to parenteral therapy is satisfactory, high dose oral therapy may be stituted after a few days e.g. Amoxycillin 500mg to 1g orally 8 hourly or to bramphenicol
0.5	g-1g orally 6 hours for patients on chloramphenicol IV.
	Severe Disease
In ac	dults, severe pneumonia should be suspected if the following features are present;
	Respiratory rate > 30 per minute
	$PaO_2 < 60 \text{mmHg or } SaO2 < 90\% \text{ on room air}$
	PaCO ₂ >50mmHg on room air
- (Chest X-ray evidence of bilateral involvement or involvement of multiple lobes
-]	Increase in size of chest X-ray opacity by 50% or more within 48 hours of admission.
- 1	Requirement for mechanical ventilation or inspired oxygen
	>35% to maintain SaO2> 90%
	Haemodynamic compromise:
	Systolic blood pressure < 90mmHg
	Diastolic blood pressure < 60mmHg
	Recent deterioration in renal function White blood cell count (4 or > 20 y 10 ⁹ /J
	White blood cell count <4 or $>30 \times 10^9/L$

☐ Penicillin G 1200mg (2 mega unit) IV 6 hourly **PLUS**

Empirical:

 □ Cloxacillin 2g IV 6 hours PLUS □ Gentamicin 4-6 mg/Kg IV once daily (maintenance dose adjusted according to renal function).
f severe or no resonse then the following may be added Erythromycin .500mg 6 hourly
f hypersensitive to penicillin ☐ Ceftriaxone 2g IV daily PLUS ☐ Erythromicin 0.5-1g IV (slow infusion over 1 hour) 6 hourly
Definitive therapy should be instituted based on bacteriological data.
If Streptococcus anginosus is proven ☐ Penicillin G 3-4mega (1800-2400mg) IV 6 hourly x 21 days If Pseudomonas aeruginosa is proven ☐ Piperacillin 4g IV 6 hourly PLUS ☐ Gentamicin 240mg IV once daily (to be adjusted for reanl function) If Staphylococcus, aureus is proven ☐ Cloxacillin 2g IV 6 hourly for 3 to 4 weeks (oral therapy can be substituted once
patient's condition is stabilized)
patient's condition is stabilized) HOSPITAL ACQUIRED PNEUMONIA
HOSPITAL ACQUIRED PNEUMONIA Refers to pneumonia not present at the time of admission and developing in patients after 48 hours of hospitalization. It is usually due to Gram-ve organisms but Staphyococcus nureus is not uncommon in Tuvalu. □ Cloxacillin 1g IV 6 hourly for 14-21 days PLUS □ Gentamicin 240mg IV once daily for 14-21 days (maintenance dose adjusted for renal function) PLUS
HOSPITAL ACQUIRED PNEUMONIA Refers to pneumonia not present at the time of admission and developing in patients after 48 hours of hospitalization. It is usually due to Gram-ve organisms but Staphyococcus nureus is not uncommon in Tuvalu. Cloxacillin 1g IV 6 hourly for 14-21 days PLUS Gentamicin 240mg IV once daily for 14-21 days (maintenance dose adjusted for renal function) PLUS Metronidazole 400mg PO 8 hourly or PR 500mg 12 hourly x 14-21 days. NEUTROPENIC PATIENTS/PATIENTS WITH RESPIRATORY DEVICE OR

IMMUNOSUPPESSED PATIENTS

therapy is 14-21 days.

Pneumonia is these patients may be recurrent and due to unusual organisms. A microbiologist or physician should be consulted regarding diagnosis and treatment. REFER to PCP under opportunistic infections HIV

ASPIRATION PNEUMONIA

Minor debris of aspiration pneumonia does not require antibiotic therapy. For severe disease or abscess formation, more prolonged and high dose treatment is indicated.
☐ <i>Streptococcus anginosus</i> , anaerobes (2 mega units) IV 4-6 hourly for 10-14 days. PLUS
☐ Metronidazole 400mg orally 8 hours for 10-14 days.
Alternative for penicillin hypersensitive patients. ☐ Chloramphenicol 500mg – 1g IV / orally 6 hourly.
Note: In addition, flucloxacillin or gentamicin may be required if infection with either staphylococci or aerobic gram negative bacteria suspected or proven. If <i>Streptococcus anginosus</i> is isolated, high dose penicillin will be required and for a longer duration, usually 21 days

Suppurative lung disease

Bronchiectasis

Introduction

Bronchiectasis is a disease characterised morphologically by the permanent dilation of bronchi and bronchioles, and clinically by recurrent or persistent bronchial infection and cough.

Most patients with bronchiectasis have a chronic cough with sputum production. The sputum is usually purulent and may be bloodstained Exacerbations of bronchiectasis are related to retained inflammatory secretions and bronchial sepsis. The condition is categorised according to the radiological appearance of the airways.

Bronchiectasis can present as a local process in one lobe or segment, or as a generalised process in both lungs. Childhood pneumonia is probably the most common cause. When focal disease is present, the cause may be intraluminal (eg foreign body, broncholith or endobronchial tumour), or due to extrinsic compression of the airway by enlarged lymph nodes.

Management

General measures

Although it is generally accepted that keeping the airways as free of secretions as possible is an important part of the management, hard evidence that it makes a difference is difficult to find. Patients ideally should be referred to a physiotherapist experienced in the area, so that an appropriate routine may be developed. This may include regular postural drainage (see figure 4) with or without percussion, advice about coughing techniques. .

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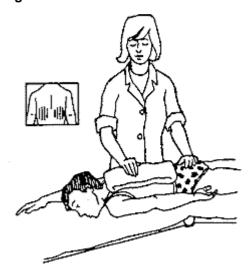
Figure 5. Postural drainage



Left lower lobe



Right lower lobe



Lower lobes, posterior segments

Include arrow to show that the table is tilted
For patients with significant airflow obstruction, nebulised bronchodilators may assist with clearing secretions.
GIVE ☐ salbutamol 100 to 200 micrograms MDI by inhalation, up to 4 times daily
OR
\Box ipratropium bromide 40 to 80 micrograms by inhalation, up to 4 times daily.
If the response to initial single-agent therapy is unsatisfactory or the patient has moderate to severe disease,
GIVE
\square salbutamol 100 to 200 micrograms MDI by inhalation, up to 4 times daily
PLUS
\square ipratropium bromide 40 to 80 micrograms by inhalation, up to 4 times daily.
Patients with poor inhalation technique can use a large-volume spacer; this improves lung deposition of the aerosol
In patients who are unable to use an MDI (with or without a spacer), USE
☐ Salbutamol 4mg orally up to 8 hourly as tolerated OR
☐ Theophylline SR 300mg bd
☐ Theophymne SK 500mg od
The effect of these agents may be monitored by self reported symptoms or by PEF or spirometry

INFECTION

If sputum becomes infected it is important to give antibiotics as early as possible GIVE

(a) Mild Disease
☐ Amoxycillin 500mg orally 8 hours for 7-10days.
Alternative:
☐ Cefaclor SR* 375mg 12 hourly for 7-10 days (for inpatient use only).
(b) Severe Disease
☐ Chloramphenicol 500mg 6 hourly orally / IV for 7-10 days
Alternative:
☐ Amoxycillin 500mg orally 8 hours for 7-10 days PLUS
☐ Metronidazole 400mg orally 8 hours for 7-10 days OR
☐ Cefactor 375mg 12 hours for 7-10 days (as a single agent) OR
☐ Doxycycline 100mg 12 hourly for 7-10 days (as a single agent)

Lung abscess

Introduction and causes

Lung abscesses usually develop either as a result of the aspiration of organisms in patients with dental caries, or as a consequence of severe necrotising pneumonia. Patients with altered conscious states (eg from anaesthesia, or alcohol intoxication, or postictal) and/or with swallowing difficulties are at particular risk. Septic emboli are occasionally a cause in intravenous drug users, often with right-sided endocarditis..

Management

The treatment of a lung abscess requires adequate drainage of the infected material and appropriate antibiotics. Where possible, attempts should be made to identify the causal organism. If there is a possibility that the patient may have aspirated a foreign body (eg a tooth, a peanut), then bronchoscopy is appropriate. If the abscess has clearly cavitated and the patient has a productive cough, the abscess is probably draining into the airways, and antibiotics and physiotherapy should be sufficient.

GIVE

Penicillin G 2 mega units IV 6 hourly PLUS
Cloxacillin 2g IV 6 hourly PLUS
Metronidazole 400mg orally 8 hours

Gentamicin should be added if aspiration is likely cause of the abscess.

^{*} Cefaclor SR 375mg not available in Tuvalu EML

Management of parapneumonic effusion and empysema

Introduction

Pleural effusion may complicate up to 50% of cases of pneumonia, and if not detected and managed appropriately may develop into a thoracic empyema. If there is clinical suspicion of a parapneumonic effusion, this should be confirmed by chest X-ray, and the fluid sampled. The fluid should be cultured. If possible, the pleural fluid pH should be assessed on a heparinised sample and fluid should be sent for routine biochemistry including LDH.

Drainage

As with any collection of pus, adequate drainage is the most important aspect of management.

Adequate drainage of empyema is essential. The duration of therapy is usually prolonged.

G	IVE
	Penicillin G 2 mega units IV 6 hourly PLUS
	Cloxacillin 2g IV 6 hourly PLUS

Interstitial Lung Disease

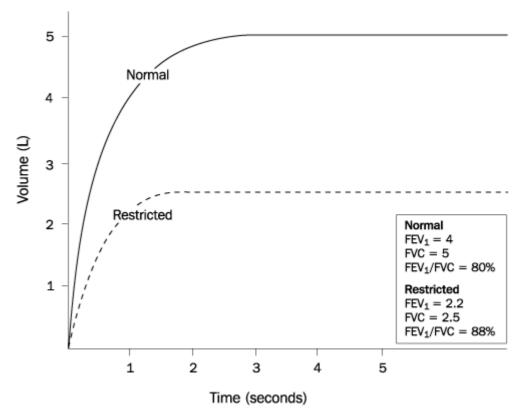
☐ Metronidazole 400mg orally 8 hours

Introduction

Many different pathological processes can affect the lung interstitium. In addition to infection by viruses, bacteria and fungi (the pneumonias), cardiogenic oedema, and fibrotic reaction to accumulation of nonorganic dust particles (the pneumoconioses), there are also many conditions in which the lung interstitium becomes inflamed, infiltrated or fibrosed

They are characterised by a restrictive pattern on spirometry testing

Spirogram showing restrictive ventilatory defect



Cryptogenic fibrosing alveolitis (idiopathic pulmonary fibrosis)

Cryptogenic fibrosing alveolitis or idiopathic pulmonary fibrosis is a specific form of chronic fibrosing interstitial pneumonia limited to the lung. Cryptogenic fibrosing alveolitis is the most common nonspecific interstitial lung disease of unknown aetiology in the elderly population, although it can occur at any age It has a peak age of onset in the 50s and 60s. and a smoking history is common. The pathology is that of 'usual interstitial pneumonia' with patchy foci of fibroproliferation. The usual prognosis is poor, with death occurring approximately 2 years after diagnosis.

The diagnosis is dependent upon:

	a typical history of slowly progressive breathlessness over months to years
	basal crackles on chest auscultation
	typical basal and peripheral reticular shadowing on XRay or computerised tomography
	of the lungs
	typical restrictive lung function abnormalities.
Th	e best evidence is for an attempt at stabilisation and improvement in survival with a
cor	mbination of oral prednisolone (low-dose) and oral azathioprine. Use

prednisolone 0.5 mg/kg lean body weight (LBW) orally, daily for 4 weeks; then 0.25 mg/kg LBW daily for 8 weeks; then 0.125 mg/kg LBW daily thereafter (ie approximately 30 to 40 mg/day tapered to 7.5 to 10 mg/day)

PLUS

azathioprine 50 mg orally, daily, increasing by 25 mg every 1 to 2 weeks) up to a maximum of 150 mg (approximately 2 to 3 mg/kg LBW) orally, daily.

This should only be added if frequent monitoring of Wbc, platelets and liver function tests is possible

If this regimen is used, a therapeutic trial of at least 3 months is necessary. If the patient continues to deteriorate on clinical and/or physiological criteria, consideration should be given to stopping medication.

Other interstitial pneumonias

Interstitial pneumonias, other than cryptogenic fibrosing alveolitis, include the following:
☐ desquamative interstitial pneumonia (DIP)
□ nonspecific interstitial pneumonia (NSIP)
☐ organising pneumonia (OP)—previously known as 'bronchiolitis obliterans organising
pneumonia (BOOP)' or 'chronic organising pneumonia (COP)'.

These interstitial lung diseases were formerly included under the broad heading of cryptogenic fibrosing alveolitis/idiopathic pulmonary fibrosis, but are now recognised as separate conditions, with individual pathological, clinical and radiological characteristics. All have a better prognosis than cryptogenic fibrosing alveolitis, related to corticosteroid responsiveness. Treatment should commenced with

prednisolone 30 to 50 mg orally, daily, with duration and subsequent tapering dependent on clinical, physiological and radiological response.

Sarcoidosis

Sarcoidosis is a multisystem granulomatous disease. The mediastinal lymph nodes or lungs are affected in more than 90% of cases. Overall, the prognosis in sarcoidosis is good, with at least 50% of pulmonary abnormalities eventually showing complete radiological clearing. The best outlook is in young patients presenting with subacute symptoms, erythema nodosum and bilateral hilar lymphadenopathy. Prognosis is worse in middle-aged patients presenting with an insidious onset of sarcoidosis, diffuse lung infiltration and pulmonary function abnormalities. Hypercalciuria is very common; however, frank hypercalcaemia occurs only occasionally

Corticosteroids usually improve systemic symptoms, pulmonary function and radiological appearances. BUT

Acute or subacute sarcoidosis with bilateral hilar lymphadenopathy is				
likely to settle spontaneously and corticosteroids are seldom required.				
☐ Musculoskeletal pains and erythema nodosum should be controlled with				
nonsteroidal anti-inflammatory drugs (NSAIDs).				

☐ Corticosteroids are indicated if pulmonary infiltrates are associated with breathlessness and significantly impaired pulmonary function or if pulmonary function is worsening over time.
☐ Hypercalcaemia should be treated with corticosteroids in addition to dietary control of calcium and vitamin D intake and high fluid intake.
 ☐ Uveitis normally requires corticosteroids, either topically or systemically, or both. ☐ Central nervous system, cardiac, or other severe extrathoracic organ involvement,
eg hepatitis, should be treated with corticosteroids.
If corticosteroids are to be given, use
prednisolone 20 to 40 mg orally, daily for 6 to 8 weeks.
If there is no response after 6 to 8 weeks, taper the dose to zero.
If there is a response, taper the dose to 10 to 15 mg orally, daily as a maintenance dose for 6 to 12 months.
Illness may present as acute or subacute episodes of pyrexia, chills and malaise with shortness >.
Drug-induced interstitial lung disease Eosinophilic reactions
Lung parenchymal interstitial eosinophilic infiltration gives breathlessness and sometimes a cough; the patient may also wheeze (suggesting an airway component as well). A maculopapular rash occurs frequently. There may be pyrexia. An immunological reaction is the likely cause.
Drugs that may be implicated include:
☐ antibiotics (, nitrofurantoin, penicillins, sulfonamides (including co-trimoxazole), tetracyclines)
 □ anti-inflammatory drugs (aspirin, sulfasalazine) □ cytotoxic drugs (methotrexate)
 □ antipsychotics and antidepressants (chlorpromazine,) □ anticonvulsants (carbamazepine, phenytoin).
For treatment, removal of the drug is paramount.
In severe or moderately severe cases, judged on clinical criteria, a short course of prednisolone can be given; use
prednisolone 20 to 40 mg orally, daily for 2 weeks.

Scuba diving

People with any significant obstructive airways disease including astma and COPD should be automatically disqualified from scuba diving. This is because of the theoretical risk of localised gas trapping, due to airway narrowing, or the presence of bullae, giving an increased risk of barotrauma. Individuals with wheeze precipitated by exercise or cold should be advised not to dive.

Any history of spontaneous pneumothorax precludes scuba diving because of the almost certain presence of bullae or blebs on the visceral pleura. Lung bullae and history of pneumothorax increase the risk of barotrauma and are a contraindication to diving.

Decompression

Divers with or without respiratory disorders may develop decompression sickness (the bends) and need decompression in a hyperbaric oxygen chamber. They should be transported for treatment as a matter of urgency.

The only one close to Tuvalu is in Fiji, at the Suva Private Hospital

Address and tel phone number to be inserted

Pregnancy and breastfeeding

Pregnancy and respiratory drugs

The major period of danger for teratogenic effects of drugs 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.

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 the need for plasma concentrations liable to have a pharmacological effect on the fetus.

Attacks of asthma during pregnancy may reduce the amount of oxygen available to the fetus, so it is particularly important that asthma is well controlled. If this is achieved, asthma has no important effects on pregnancy, labour or the fetus. Severe exacerbations should be treated promptly with conventional therapy. Most asthma medications are safe to use during pregnancy.

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 breast milk 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 breast milk.

For these reasons the time of dosing in relation to breast feeding does not make much difference.

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 breast milk.

Respiratory drugs in pregnancy and breastfeeding

Drug	Status in Pregnancy	Use in breastfeeding
Aminophyl line	Safe	use with caution; monitor infant for irritability
beclomethasone dipropionate	Can be used because its inhaled	May be safe to use
Dexamethasone	Safe	Safe
Hydrocortisone	Safe	Safe
Ipratropium bromide	safe because inhaled	Safe
Prednisolone	Safe	safe
Promethazine	may cause foetal drowsiness	safe in single dose
Salbutamol	Safe	Safe
Theophylline	crosses placenta in significant amounts. Avoid if possible	use with caution; monitor infant for irritability

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