Case 3 A 31-year-old woman with poorly-controlled asthma

Frances Johnson is a 31-year-old woman who presents to her GP with mild shortness of breath and a non-productive cough, worse at night. Her symptoms deteriorated slightly with a cold several weeks previously and have not improved. She also complains of itchy eyes, sneezing and persistent blockage of her nose.

She was diagnosed with asthma at the age of 12 and given a blue and a brown inhaler. She remembers undergoing skin prick testing, which was positive for house dust mite allergy. Her asthma was not a problem until the last few years when she restarted inhaled salbutamol. She now needs this 3–4 times per day to help her breathing. She does not measure her peak flow rates as she has lost her peak flow meter but remembers her best was around 400 L/min.

Summarize Miss Johnson's problems

- She has a past history of asthma as a child which has recurred as an adult
- Her symptoms appear to have worsened following a respiratory tract infection
- Her asthma symptoms are not controlled with 'as needed' use of a bronchodilator (salbutamol)
- Her persistent upper respiratory tract symptoms (itchy eyes, sneezing, nasal blockage) could indicate allergic rhinitis

How should you approach assessment of her asthma?

The key issues to be determined in this consultation are:

- Is she having an acute exacerbation of asthma severe enough to require oral steroid treatment or hospital referral?
- If not, how should her chronic asthma be managed?

What other information do you need to complete this assessment?

Criteria defining moderate, acute severe and life-threatening asthma exacerbations are given in Box 31. Pulse rate, respiratory rate and peak flow measurement are required to complete the assessment.

On examination:
She is speaking in full sentences
Respiratory rate is 20 breaths/min
Pulse rate is 90 beats/min
Her peak expiratory flow rate (PEFR) is 320 L/min (best 400 L/min)
She has a mild expiratory wheeze to hear on auscultation of her chest

How does this new information alter your assessment?

She has no features of an acute severe asthma attack. As her peak flow is 80% of her previous best and her symptoms are slightly worse than usual but not increasing, she does not meet the criteria for a moderate asthma exacerbation. She can be treated as having poorly controlled chronic asthma.

What are the aims of asthma treatment?

The aims of treatment are to:

- Control symptoms
- Minimize exacerbations
- Achieve best possible lung function
- Minimize side-effects and inconvenience of treatment

Asthma control is assessed against the standards given in Box 32.

What treatment will best achieve control of her asthma symptoms?

Asthma is a chronic inflammatory process of the airways (Fig. 4, p. 5 & Fig. 27, p. 47). Symptoms are caused by a combination of bronchospasm, mucosal oedema and mucus hypersecretion, causing airflow obstruction. The mainstay of treatment for asthma is **corticosteroids**, which reduce airway inflammation (Box 33) producing symptom control, preventing exacerbations and maintaining lung function.

Box 31 Levels of severity of acute asthma exacerbations in adults (British Thoracic Society Guidelines)

Moderate asthma exacerbation
- No features of acute severe asthma
- Increasing symptoms
- PEFR 50–75% predicted or best

Acute severe asthma
Any one of:
- Cannot complete sentences in one breath
- Tachypnoea (≥25 breaths/min)
- Tachycardia (≥110 beats/min)
- PEFR 33–50% of predicted or best

Life-threatening asthma
Any one of:
- Silent chest
- Cyanosis
- Feeble respiratory effort
- Bradycardia, dysrrhythmia or hypotension
- Exhaustion, confusion or coma
- PEFR <33% of predicted or best
- Arterial oxygen saturation <92%, PaO_2 <8 kPa

Near-fatal asthma
- Raised $PaCO_2$

Box 32 Features of well-controlled asthma

The British Thoracic Society Asthma Guidelines define good asthma control as:
- Minimal symptoms during day and night
- Minimal need for reliever medication
- No exacerbations
- No limitation of physical activity
- Normal lung function (in practical terms FEV_1 and/or PEFR >80% predicted or best)

KEY POINT

Treatment of symptomatic asthma must include the use of **corticosteroids** to control airway inflammation in all but the mildest cases.

Box 33 Pharmacology of corticosteroids

Mechanism of action
Inhibit inflammation by:
- Inhibiting influx of inflammatory cells
- Inhibiting release of inflammatory mediators from inflammatory cells
- Reducing microvascular leakage and oedema

Some of these actions are mediated by stimulating production of lipocortin which inhibits leukotriene and prostaglandin synthesis.

Uses
In respiratory medicine corticosteroids are used for diseases including asthma, COPD and interstitial lung diseases:
- In higher doses to treat inflammation
- In lower doses to prevent recurrence of inflammation

Pharmacokinetics
- Steroids can be given topically by inhaler (e.g. beclometasone, budesonide, fluticasone), which minimizes side-effects and maximizes activity in airways inflammation
- Higher doses given intravenously (hydrocortisone) or orally (prednisolone) may be required to treat severe inflammation, but should be given for as short a time as possible to minimize side-effects

Side-effects
Inhaled steroids
Local effects include sore mouth, oral thrush, hoarse voice

Systemic steroids
- Immune suppression – increased susceptibility to infection, especially organisms normally killed by cell-mediated immunity (e.g. *Candida*, TB, protozoa)
- Metabolic effects – e.g. osteoporosis, diabetes, hypertension, cushingoid features (moon face, buffalo hump), thin skin, easy bruising, suppress growth in children
- Adrenal suppression – hypoadrenalism if stop treatment quickly

How are corticosteroids given to asthmatic patients?

The administration of corticosteroids in asthma requires careful balancing of risks and benefits. In chronic asthma, inhaled steroids are usually sufficient to control asthma inflammation with minimal side-effects. In acute asthma, oral or intravenous corticosteroids are given as high treatment doses are required to control the asthma exacerbation and the benefits of treatment outweigh the risk of side-

effects over the short treatment period required. The pharmacology of corticosteroids is discussed further in Box 33.

What corticosteroids should Miss Johnson receive?

Treatment should follow standard stepped guidelines for the management of chronic asthma (Box 34). Key features of this approach are:

Box 34 Stepped care for chronic asthma in adults

Step 1. Occasional relief bronchodilators
Short-acting β_2-agonist as required (up to once daily)
Asthma poorly controlled ⇅ Asthma well controlled

Step 2. Regular inhaled preventer therapy
Short-acting β_2-agonist as required
Regular standard dose inhaled steroid
Asthma poorly controlled ⇅ Asthma well controlled

Step 3. Inhaled corticosteroids plus long-acting inhaled β_2-agonist
Short-acting β_2-agonist as required
Regular standard dose inhaled steroid
Regular long-acting β_2-agonist
If still not well controlled:
Increase dose of inhaled steroid to upper end of standard dose

Then try one of:
- Leukotriene receptor antagonist
- Modified-release oral theophylline
- Modified release oral β_2-agonist

Asthma poorly controlled ⇅ Asthma well controlled

Step 4. High-dose inhaled corticosteroids and regular bronchodilators
Short-acting β_2 agonist as required
Regular *high* dose inhaled steroid
Regular long-acting β_2-agonist
and
6-week sequential trial of one or more of:
- Leukotriene receptor antagonist
- Modified-release oral theophylline
- Modified release oral β_2-agonist

Asthma poorly controlled ⇅ Asthma well controlled

Step 5. Regular corticosteroid tablets
Short-acting β_2-agonist as required
Regular high-dose inhaled steroid
One or more long-acting bronchodilators
Regular prednisolone tablets as single daily dose

- The aim is to abolish symptoms and optimize peak flow as soon as possible
- The patient should be started on treatment on the stepped guideline at the level most likely to achieve this aim
- Once control of asthma is achieved treatment can be stepped down if control is good or stepped up for increasing symptoms

Miss Johnson's symptoms are not controlled by using 'as required' bronchodilators (step 1). A reasonable next step would therefore be to add in 'a regular standard dose inhaled corticosteroid' to her treatment (step 2).

Which inhaled steroid should the GP choose?

The main options are:
- Beclometasone
- Budesonide
- Fluticasone

Choice

In general, all of these drugs have similar efficacy and side-effects. Selection therefore comes down to patient choice of delivery device and cost. A reasonable first choice would be either beclometasone (metered dose inhaler; Fig. 29a) or budesonide (dry powder inhaler; Fig. 29d).

Dose

Beclometasone and budesonide have similar potency, therefore are used at a similar dose of 100–400 µg twice daily. However, fluticasone is more potent and provides equal clinical activity to beclometasone and budesonide at *half* the dosage. If used at step 2 it should therefore be given at 50–200 µg twice daily.

> *Miss Johnson's GP prescribes beclometasone 200 µg one puff morning and night and salbutamol 100 µg to use two puffs as required. The GP demonstrates use of the metered dose inhaler (MDI) and explains the difference between a reliever (bronchodilator) and a preventer (steroid) inhaler.*

What is a metered dose inhaler and how is it used?

These inhalers comprise small canisters which contain the drug and a pressurized propellant gas. When the nozzle is compressed, a puff of drug is expelled from the canister. The difficult part for the patient is to coordinate a deep breath in with the puff of drug to ensure the medication reaches the lung. Patients should be advised to use their MDI as follows:

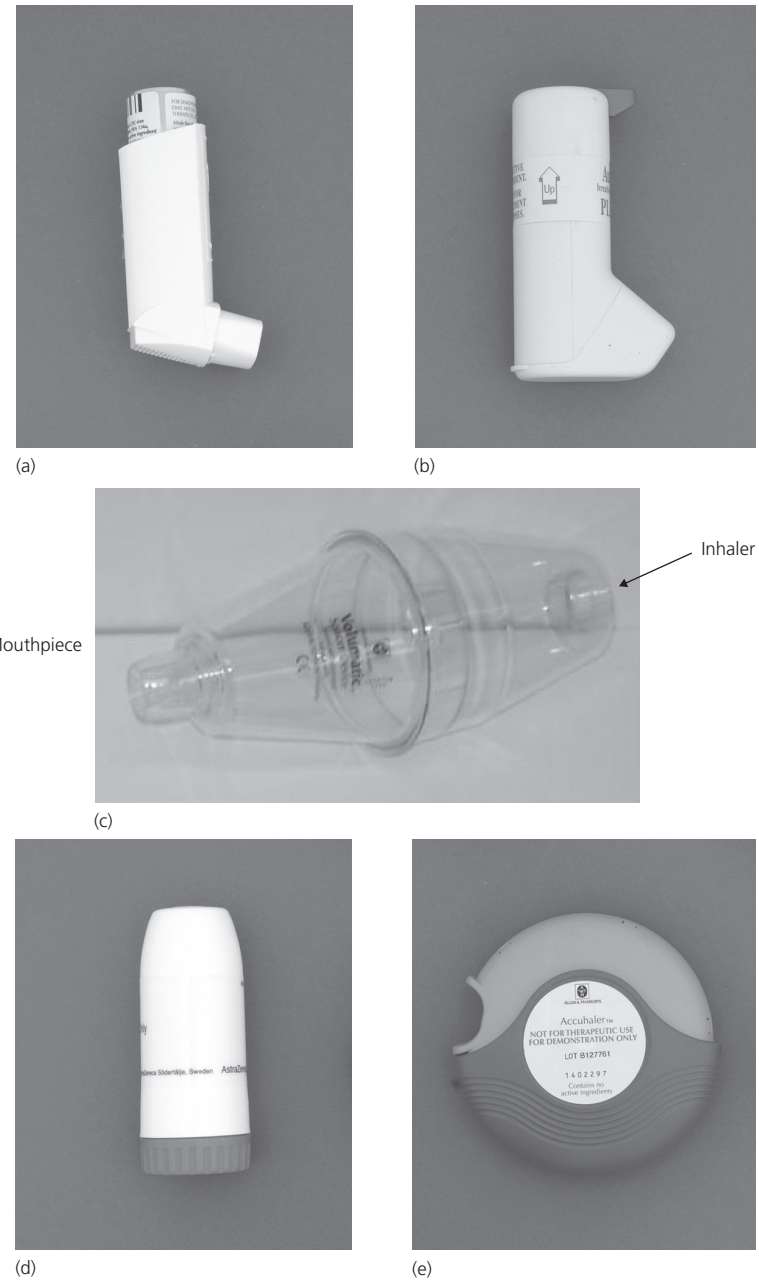

Figure 29 Different types of inhaler used in the treatment of asthma. (a) Metered dose inhaler (see text). (b) Autohaler. Similar to metered dose inhaler but release of drug initiated by inhalation and not by pressing canister. Useful for patients with arthritis or other condition that prevents them from pressing the nozzle, or for patients who have difficulty coordinating inspiration and inhaler actuation. (c) Volumatic spacer device. The mouthpiece of the metered dose inhaler is inserted into the volumatic as shown. A puff of medication is released into the volumatic and the patient inhales through the device in their own time. Use of a volumatic reduces the need for coordination of inhalation and inhaler actuation. It also results in drug deposition deeper in the airways. (d) Turbohaler. This device contains drug in dry powder form which is released by turning the inhaler base. The patient then inhales the drug in their own time. This requires less coordination than the metered dose inhaler and can be easier for patients to use. Potential disadvantages are airway irritation by the powder. (e) Accuhaler. This is an alternative dry powder inhaler.

- Remove the cap from the mouthpiece
- Shake the inhaler several times to mix the drug
- Breathe all the way out
- Put the inhaler in your mouth and seal your lips around the opening
- Simultaneously breathe in at a moderate and steady rate and depress the canister to release the aerosolized drug, optimizing drug delivery to the lungs
- Hold your breath for 10 s
- Breathe out, then repeat

How would you explain the difference between a reliever and a preventer inhaler to Miss Johnson?

It is important to explain that asthma symptoms are caused by swelling in the tubes carrying air into the lung. These

Box 35 Top tips – inhaler colours and appearance

If a patient with asthma is not sure what inhalers they are taking, the colour or appearance of the inhaler will often give the drug content away.

Inhaler	*Colour/appearance*
Short-acting β_2-agonist	Blue
Short-acting antimuscarinic	Grey or clear with some green
Long-acting β_2-agonist	Green
Long-acting antimuscarinic	Oval container requiring insertion of a capsule
Inhaled steroid	Brown/maroon Orange (fluticasone)
Combined steroid/long-acting β_2-agonist	Purple or red

swollen tubes are very sensitive and can easily go into spasm. Both the swelling and the spasm narrow the air tubes, making it difficult to breathe and causing a cough. There are two types of treatment given to patients with asthma:

1 The 'preventer', or brown inhaler (steroid) (Box 35). This reduces swelling in the air tubes and, once the airways have recovered, prevents the swelling from coming back. The preventer inhaler must be taken every day to have its effects, even if the patient is feeling well
2 The 'reliever', or blue inhaler (β_2-agonist) (Box 35). This will not get rid of the underlying swelling of the air tubes, but it will relax muscle spasm and so can be used any time to make breathing easier or reduce cough. The patient does not need to take this inhaler if they feel well, but they should carry it with them to take if symptoms develop. The effects of this inhaler last about 2–4 h and repeat doses can be taken throughout the day

Frances Johnson returns for review after 2 weeks of step 2 treatment. She reports that she has noticed some improvement but she still has a non-productive cough. She is still using her short-acting bronchodilator 2–3 times per day and her steroid inhaler twice a day. Her PEFR is 350 L/min and her chest is now clear to auscultation. She comments that her blocked nose adds to her difficulty with breathing at night.

Should her asthma treatment be changed at this stage?

Her persistent cough, bronchodilator use and suboptimal PEFR indicate that her asthma is not well controlled (Box 32). She probably needs an increase in treatment to step 3. However, before this is carried out the GP should check that:

- Her inhaler technique is optimal
- She is adhering to the treatment regime
- Asthma triggers have been minimized

If these are satisfactory and her symptoms are still not controlled then her treatment should be increased to step 3 (Box 34).

Box 36 Allergic rhinitis

Patients with atopy can develop nasal symptoms as a result of an allergic reaction in the nasal mucosa, similar to that seen in the lungs with asthma (Fig. 27).

Seasonal rhinitis

This is also known as hayfever and is the most common allergic disease with the highest prevalence in the second decade of life. Symptoms include:

- Nasal blockage, watery discharge and sneezing
- Itchy eyes, soft palate and ears
- Watering eyes

Seasonal rhinitis is limited to the part of the year when pollen and spore allergens are at high concentrations in the environment. Allergens causing seasonal rhinitis include:

- Tree pollens – in April and May
- Grass pollens – from June to August
- Mould spores – from July to September

Perennial rhinitis

This is most common in the second and third decades of life. Symptoms are predominantly nasal blockage and discharge with loss of sense of smell and taste. Allergens causing perennial rhinitis include:

- House dust mites
- Domestic pets
- Moulds

Chronic rhinitis may lead to the development of *nasal polyps*, which are overgrowths of the nasal or sinus mucosa that remain attached to their origins by a thin stalk. These can obstruct the nasal passages, causing loss of sense of smell and taste and mouth breathing.

How do you account for her nasal symptoms?

Nasal obstruction in patients with asthma may be caused by allergic rhinitis (Box 36) or nasal polyps. In her initial presentation Miss Johnson complained of itchy eyes and

sneezing, making allergic rhinitis the likely diagnosis. Allergic rhinitis can be differentiated from infective rhinitis (the common cold) by persistence of symptoms for several weeks.

How would you confirm your provisional diagnosis of allergic rhinitis?

The diagnosis of allergic rhinitis is based on a detailed history of symptoms, atopy and nasal reactions to allergen exposure and removal. Nasal inspection should be performed to look for polyps. Investigations are not usually indicated but skin prick testing can be used to confirm allergic reactions to specific allergens that may guide allergen avoidance strategies.

How would you treat her rhinitis?

There is some evidence that treatment of rhinitis may improve asthma outcomes, as well as relieving nasal symptoms. Miss Johnson's GP should therefore consider treating her rhinitis in parallel with her asthma treatment. Treatment options for allergic rhinitis include antihistamines, decongestants and corticosteroids (Box 37).

> *The GP finds that Miss Johnson's asthma trigger factors have been addressed satisfactorily. She has no household pets, she does not smoke and she lives in a house with wooden floors and window blinds. She has mattress and pillow covers to reduce house dust mite exposure. Her inhaler technique is satisfactory and she is adamant that she is taking the prescribed treatment. Her GP escalates her treatment to step 3 by adding in salmeterol at a dosage of 50 µg twice a day. The inhaled steroid dosage is left at 400 µg/day. Miss Johnson is also prescribed mometasone nasal spray 50 µg (1 spray) to each nostril daily and cetirizine 10 mg in the morning for her rhinitis.*

Are long-acting β_2-agonists effective and safe in asthma?

A large number of clinical trials have investigated the use of long-acting β_2-agonists in the treatment of patients with asthma and these have recently been summarized in a systematic Cochrane review (*Cochrane Database Systematic Review* 2007 Jan 24; 1: CD001385). The review found that there were benefits for patients of using long-acting β_2-agonists (Box 38):

- Improved lung function
- Significantly fewer symptoms
- Less use of rescue medications
- Higher quality of life scores

However, the review also found that there were risks of treatment. The review estimated that for every 1250 patients with asthma treated with long-acting β_2-agonists

Box 37 Pharmacology of drugs used to treat allergic rhinitis

Drugs in each of these classes are available over-the-counter for self-medication.

Antihistamines

For example, chlorphenamine (Piriton), cetirizine:

- *Mechanism of action*. Block histamine receptors, preventing the early phase reaction of the allergic response (Fig. 27)
- *Uses.* Control symptoms in mild allergic rhinitis
- *Pharmacokinetics.* Can be given orally or topically
- *Side-effects.* Chlorphenamine causes drowsiness. Second-generation drugs (e.g. cetirizine) do not cross the blood–brain barrier and are therefore non-sedating

Decongestants

For example, ephedrine hydrochloride

- *Mechanism of action.* Sympathomimetics cause constriction of mucosal blood vessels, reducing oedema of the nasal mucosa and relieving nasal obstruction
- *Uses.* Short term to relieve congestion and allow penetration of nasal steroid
- *Pharmacokinetics*. Can be given orally or topically
- *Side-effects.* Once the use of decongestants is stopped there is secondary vasodilatation causing rebound nasal congestion and recurrence of symptoms. This is particularly problematic if decongestants are used for more than 7 days. Therefore, they should only be given as a short course

Corticosteroids

For example, beclometasone, mometasone

- *Mechanism of action.* Anti-inflammatory (Box 33)
- *Uses.* Treatment and prevention of allergic rhinitis
- *Pharmacokinetics.* Used topically in allergic rhinitis as nasal drops or spray
- *Side-effects:*
  - worsening of untreated nasal infection
  - dryness, irritation and bleeding of nasal mucosa
  - raised intraocular pressure
  - headache

Box 38 Pharmacology of β_2-adrenoceptor agonists

Mechanism of action

Act at β_2-adrenoceptors to stimulate bronchial smooth muscle relaxation through activation of adenyl cyclase

Uses

Relief of symptoms from airflow obstruction (asthma and COPD):

- Short-acting β_2-agonists (salbutamol, terbutaline) are used for acute relief of bronchospasm or to prevent bronchospasm (e.g. before exercise or going out into cold air)
- Long-acting β_2-agonists (salmeterol, formoterol) are used to maintain bronchodilatation and prevent symptoms over a 12-h period (e.g. overnight)

Pharmacokinetics

Duration of action:

- Short-acting β_2-agonists (e.g. salbutamol, terbutaline) act immediately and have a biological half-life of 2–3 h
- Long-acting β_2-agonists (e.g. salmeterol, formoterol) remain at the receptor longer and have a longer duration of action of up to 15 h. For example, salmeterol has a long lipophilic tail which binds to an exoreceptor near the β_2-adrenoceptors in airway smooth muscle

Route of administration:

- Inhaled directly into the lungs – maximizes efficacy and minimizes side-effects
- Short-acting β_2-agonists can also be given:
  - by nebulizer – higher dose and less coordination and respiratory effort required to take therapy
  - orally – if inhalers cannot be used (e.g. small children)
  - intravenously – emergency use where severe airflow obstruction prevents inhalation

Side-effects

Tremor, tachycardia, anxiety, hypokalaemia.

there was one extra asthma-related death. This was particularly seen in patients *not* taking inhaled corticosteroids.

Why might long-acting β_2-agonists increase the risk of asthma-related death?

The mechanisms are not fully understood. It is possible that long-acting β_2-agonists mask asthma symptoms and delay patients from seeking medical care, which may result in a more severe or life-threatening asthma attack. Alternatively long-acting β_2-agonists may reduce the sensitivity of β_2-receptors, making short-acting β_2-agonists less effective during an acute attack.

What are the implications of these findings for patient care?

Long-acting β_2-agonists should *not* be used in patients with asthma without inhaled corticosteroids. However, they appear to have clinical benefit when used with inhaled corticosteroids as per stepped guidelines.

KEY POINT

Long-acting β_2-agonists should not be prescribed to patients with asthma without inhaled corticosteroids.

Miss Johnson returns for review 2 weeks later and has improved again. Her cough is much better and she has used her short-acting bronchodilator inhaler once in the last week. Her PEFR is now 380 L/min and her nasal symptoms are improving. Her GP refers her to the respiratory nurse for a self-management plan. Miss Johnson tells the nurse that she is not happy about taking inhaled steroids as she is worried she may put on weight and asks how serious it would be if she stopped taking them.

What is an asthma self-management plan?

This is a personalized action plan usually compiled by a respiratory nurse in consultation with the patient. It gives the patient instructions on how to increase treatment if their symptoms worsen or if the PEFR drops below a level identified as a percentage of their best PEFR. Similarly, if the patient's symptoms have been controlled and stable for suitable time period (e.g. 3 months), instructions are given about how to step down treatment. The patient is also given a reserve course of antibiotics and steroids so that prompt treatment can be started for an infective exacerbation. The management plan empowers the patient to manage their asthma themselves with the support of the respiratory nurse, who is usually contactable at short notice.

Box 39 Pharmacology of leukotriene receptor antagonists (montelukast, zafirlukast)

Mechanism of action

The cysteinyl-leukotrienes (LTC_4 and LTD_4) are inflammatory mediators released by eosinophils and other inflammatory cells during the pathogenesis of asthma. They stimulate bronchoconstriction, mucus hypersecretion and mucosal oedema. Montelukast and zafirlukast block leukotriene receptors, inhibiting these inflammatory effects.

Uses

Step 3 or 4 treatment to control chronic asthma. Appear to be particularly effective in exercise- and aspirin-induced asthma and patients with associated rhinitis and/or nasal polyps.

Pharmacokinetics

Unlike most other asthma drugs they are taken orally once per day which may aid adherence to treatment.

Side-effects

These include gastrointestinal disturbances, headache, thirst and insomnia.

Box 40 Pharmacology of xanthines (theophylline, aminophylline)

Mechanism of action

Xanthines inhibit phosphodiesterase enzymes which metabolize secondary messengers inside airway smooth muscle cells. Phosphodiesterase enzyme inhibition causes a build-up of cyclic adenosine monophosphate (cAMP) in airway smooth muscle cells and bronchodilatation.

Uses

Bronchodilatation in patients with severe acute asthma or stable COPD.

Pharmacokinetics

- Xanthines have a narrow therapeutic range; their lowest effective plasma concentration (10 μg/mL) is close to their toxic concentration (20 μg/mL)
- Xanthines are metabolized in the liver by cytochrome p450 enzymes. If drugs that inhibit these enzymes are co-prescribed with xanthines, xanthine plasma levels may rise from therapeutic to toxic concentrations. Liver enzyme inhibitors include:
  - erythromycin
  - ciprofloxacin
  - sodium valproate
- Plasma levels of xanthines should be monitored routinely and the dose reduced if liver enzyme inhibitors are co-administered

Side-effects

More likely at toxic plasma concentrations:

- Nausea and vomiting
- Arrhythmias and convulsions

What therapeutic options could have been considered if Miss Johnson's asthma had not improved with step 3 treatment?

The next treatment options (Box 34) would have been to optimize step 3 treatment by increasing the dose of inhaled steroids and, if asthma was still not well controlled, to try a leukotriene antagonist (Box 39), theophylline (Box 40) or an oral β_2-agonist. If asthma still was not well controlled, step 4 options include high-dose inhaled corticosteroids and step 5 involves commencement of regular oral prednisolone.

How should the nurse answer Miss Johnson's question?

The nurse should explain that it is essential that Miss Johnson's asthma is adequately treated as if not:

- She may have an acute exacerbation which could be life-threatening
- Chronic inflammation can lead to airway remodelling with permanent narrowing and disabling breathlessness in the long term

The nurse can reassure Miss Johnson that very little of the inhaled steroids will be absorbed and she is extremely unlikely to put on weight. However, she should advise her that inhaled steroids can cause oral thrush and a hoarse voice. Miss Johnson can minimize the risk of these effects by rinsing her mouth and gargling with water after taking the inhaler. Miss Johnson can be reassured that it may be possible to reduce her inhaled steroids in the future if her symptoms are controlled.

Outcome. Miss Johnson took her inhaled steroids and salmeterol regularly for the next 3 months. Once her symptoms were well controlled she stopped the salmeterol and 6 weeks later reduced inhaled steroids from 400 to 200 μg/day. Six months later she was stable and opted to stop inhaled steroids altogether. She remained aware, using her self-management plan, that she would need to restart treatment and seek advice should her symptoms recur.

CASE REVIEW

Frances Johnson presents to her GP with symptoms of asthma recurring from childhood following a disease-free period in her twenties. Her GP finds no evidence of a moderate or severe exacerbation and decides to treat her as chronic poorly controlled asthma according to standard stepped asthma care guidelines. Miss Johnson requires escalation of asthma treatment to step 2 (addition of standard dose inhaled steroids), then step 3 (addition of long-acting β_2-agonist) to control her symptoms. She also receives nasal steroids and antihistamines for rhinitis and sees the respiratory nurse for a self-management plan. After her symptoms have been controlled for some months she is able to step her treatment down, but remains aware of the need to restart treatment and seek help early if her symptoms recur.

KEY POINTS

- The aims of asthma treatment are to control symptoms, minimize exacerbations and optimize lung function
- Features of poorly controlled asthma include:
  - daytime or nocturnal symptoms
  - regular use of reliever medication
  - falling PEFR
  - recent exacerbations
- In a patient presenting with poorly controlled asthma, the first step should be to determine whether this is a moderate or severe exacerbation requiring acute treatment
- If the poor asthma control is chronic, control should be improved using the stepped guidelines for chronic asthma management
- The mainstay of asthma treatment is **corticosteroids**
- In chronic asthma, corticosteroids are given as inhalers to optimize drug delivery to the affected airways and minimize side-effects
- After taking inhaled steroids patients should be advised to rinse their mouth and gargle to reduce the risk of oral thrush or a hoarse voice
- The stepped guidelines should be used both to increase treatment in patients with poorly controlled asthma and to reduce treatment in patients with well-controlled asthma. Treatment should be reviewed and adjusted approximately every 3 months
- Long-acting β_2-agonists can be used in the treatment of asthma, but should *only* be used with inhaled steroids as long-acting β_2-agonists are associated with an increased risk of asthma-related deaths if used alone
- Patients with asthma commonly have associated allergic rhinitis, which should be treated as this may improve symptoms and assist with asthma control
- Patients with asthma should see a nurse with appropriate expertise to develop a self-management plan and for support in management of their condition