

- lightheadedness, faintness or dizziness
- breathlessness
- palpitations
- sweating
- dry mouth with aerophagy
- agitation
- fatigue and malaise

Other symptoms include paraesthesia of the extremities, peri-oral paraesthesia and carpopedal spasm.

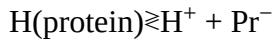
Carpopedal spasm: biochemical explanation

CO_2 loss from hyperventilation



$\text{pCO}_2 \downarrow \rightarrow \text{HCO}_3^- \downarrow$ and $\text{pH} \uparrow$ (respiratory alkalosis)

H^+ depleted and replenished from plasma proteins



Protein anions accumulate and take up calcium



Thus ionised calcium is depleted causing hypocalcaemic tetany.

Management

- Reassure.
- Encourage patients to identify the cause and then control their rate and depth of breathing.

⌚ Adjustment disorder with anxious mood

This term is reserved for patients who present with anxiety symptoms within 3 months of response to an identifiable psychosocial stressor. It is a common presentation of anxiety symptoms and should be regarded as a separate entity to a generalised anxiety disorder. It impairs social or occupational functioning.

The symptoms are in excess of the normal expected reaction to the stressor but have persisted for less than 6 months following the removal of the stressor.

The basic treatment is non-pharmacological: counselling, relaxation and stress management. A short-term course of drug treatment, e.g. diazepam for 2 weeks, can be used in severe or persisting cases.

Somatic symptom disorder⁴

Somatic symptom disorder (ssD) is defined as the tendency to experience, conceptualise and communicate mental states and distress as physical symptoms or altered bodily function. It is associated with excessive illness, worry and abnormal illness behaviour. ssD is persistent with a history of numerous unsubstantiated physical complaints over several years, beginning before the age of 30. Previously called somatisation disorder in the DSM-IV-TR, or hysteria in the past, ssD has two subtypes: those with predominantly somatic complaints and those with predominantly pain issues (previously known as pain disorder).

Symptoms include:

- gastrointestinal—nausea, vomiting, abdominal pain
- genital/sexual—dysmenorrhoea, dyspareunia, genital pain, anorgasmia
- cardiovascular—palpitations, shortness of breath, chest pain
- pseudoneurological—amnesia, loss of voice, dizziness, difficulty walking/talking/swallowing
- pain—diffuse, neck/back ache, joint/limb pain, headaches
- other—fatigue, globus, fainting

None of these symptoms has an adequate physical explanation. ssD is more common in Page 848 females. There is persistent refusal to be reassured that there is no explanation for the symptoms. There is associated impaired social, occupational and family functioning.

Management involves skilful counselling, explanation for symptoms, searching for and treating comorbid conditions (e.g. depression, anxiety) and CBT. It is preferable to be managed by a single supportive doctor. It is not malingering.

Acute stress disorder

This is defined as a constellation of abnormal anxiety-related symptoms lasting at least 3 days and occurring within 4 weeks of a traumatic event. The symptoms can include a sense of numbing, altered sense of reality, amnesia of the event, intrusive memories or dreams of the event, dissociative reactions, physiological reactions to triggers, avoidance of reminders, sleep disturbance, hypervigilance, anger and aggression, exaggerated startle response and agitation. It is appropriate to provide people with an acute stress reaction with debriefing and counselling (if

agreeable); pharmacological intervention is rarely indicated.

Anxiety in children

Anxiety disorders can occur in childhood and, if left untreated, may persist into adolescence and adulthood. Panic attacks are not uncommon. Other disorders include GAD, social phobia, obsessive-compulsive disorder, PTSD, selective mutism and separation anxiety disorder. Non-pharmacological approaches are preferred, and may require input from an educational and developmental psychologist. Separation anxiety disorder for real, threatened or imagined separation is the most common anxiety disorder; if severe and persistent, treatment (under specialist supervision) with one of the SSRIs could be considered.

When to refer

- If the diagnosis is doubtful
- If drug and alcohol dependence or withdrawal complicate the management
- If psychosis appears to be involved
- Failure of response to basic treatment
- Hospitalisation is indicated

Note: The threshold for referral varies depending on the GP's confidence and experience in dealing with anxiety, and the available referral pathways.

Practice tips

- Be careful not to confuse depression with anxiety.
- A depressive disorder can be the cause of anxiety symptoms.
- For anxiety, especially with cardiovascular symptoms (palpitations and/or flushing), always consider the possibility of hyperthyroidism and order thyroid function tests.
- Always try non-pharmacological measures to manage anxiety whenever possible.
- Be careful with the use of benzodiazepines: aim for short-term treatment only.

Tips to beat stress

- Get enough sleep and rest.
- Listen to music.
- Do things that you enjoy.
- Look at positives.
- Develop strategies to laugh.
- Go to the movies or a show weekly.
- Consider getting a pet.
- Remember that your job is what you do (not who you are).
- Have regular chats with close friends.
- Exercise for 30 minutes, 4 or 5 times a week. There is good evidence this helps.
- Learn to meditate.
- Avoid interpersonal conflicts.
- Learn to accept what you cannot change.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Anxiety disorder
- Coping with a crisis
- Phobias
- Post-traumatic stress disorder
- Social phobia
- Stress: coping with stress

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71 Difficult behaviours

There are patients in every practice who give the doctor and staff a feeling of ‘heartsink’ every time they consult.

THOMAS O'DOWD 1988¹

Difficult, demanding and angry patients

Weston defines a ‘difficult patient’ as one with whom the physician has trouble forming an effective working relationship.² However, it is more appropriate to refer to difficult problems rather than difficult patients—it is the patients who have the problems while doctors have the difficulties.

The proportion of consultations that are taken up by *difficult patients* (also called *heartsink* or *hateful* patients) has been measured as being 15%.² While in the minority, by their nature they can often take up a disproportionate amount of the doctor’s time, energy and emotional reserves. One difficult patient may disrupt an entire consulting session. The concept of the difficult patient was first popularised by the landmark paper by Groves in 1978,³ and many difficult patient types have since been described. Four of the more common and better-known types⁴ are as follows:

1. *The dependent clinger*

Dependent clingers require constant reassurance, and have an unquenchable need for explanation, affection and attention. They may break social or professional barriers to meet this need, such as calling the doctor at home or continually making unplanned presentations at the surgery. The doctor can feel threatened by such patients, and if pushed away, dependent clingers can feel rejected, which may exacerbate their behaviour. They respond well to an empathic approach that needs to be delivered within clearly defined and enforced boundaries.

2. *The entitled demander*

Entitled demanders attempt to control the doctor through intimidation and by inducing guilt or fear in the doctor. They project an air of superiority and entitlement, and may demand tests or consultation prioritisation, withhold payment and are often litigious. The doctor may understandably feel afraid and despairing in such situations, but this type of difficult patient is

often driven by an underlying insecurity and is attempting to obtain control through bluster. The appropriate use of power is clearly required for such patients, but it is important (and often difficult) to stay in control and interact in a respectful and non-confrontational manner. This may include pointing out calmly but clearly when boundaries are being crossed.

3. *The manipulative help rejecter*

Manipulative help rejecters are patients who are on a self-destructive path but refuse to take important medical advice. They crave the relationship with the doctor, and solving or improving the medical situation may threaten that relationship. Substance abuse is a common manifestation of how manipulative help rejecters present and manipulate the relationship, as are non-compliance and chronic pain issues. The doctor can feel frustrated and even demoralised, and it is important to reflect on our own feelings and expectations with such patients.

4. *The self-destructive denier*

Rather than wanting to cling to the doctor (like the manipulative help rejecter), self-destructive deniers appear to want to damage themselves, their motivation driven by self-loathing. They may induce feelings of indifference or hatred in the doctor because of their destructive behaviour and apparent refusal to change their ways. An empathic approach is the most useful here, but this may be emotionally draining for the doctor.

An inevitably poor consultation will follow if we allow feelings of hostility to affect our communication with the difficult patient, especially the demanding, angry or ‘compo’ patient.

However, it is important not to misdiagnose organic disease and also to consider the possibilities of various psychological disorders, which may be masked. Hahn and colleagues identified six diagnoses:^{3,5}

- generalised anxiety disorders
- multi-somatoform disorder
- dysthymia
- panic disorder
- major depression
- drug dependency/alcohol abuse

Page 851

It is therefore appropriate to maintain traditional standards by continually updating the database, integrating psychosocial aspects, carefully evaluating new symptoms, conducting an appropriate physical examination and being discriminating with investigations.

Management of the violent and dangerous patient is presented in [CHAPTER 69](#) on the disturbed patient.

Management strategies

Our professional responsibility is to rise above interpersonal conflict and facilitate productive communication by establishing a caring and responsible relationship with such patients. An appropriate strategy is to follow Professor Aldrich's precepts for the 'difficult' patients who do not have an organic disorder or a psychiatric illness:⁶

1. Give up trying to cure them—they are using their symptoms to maintain their relationship with you: accept them as they are.
2. Accept their symptoms as expressions of their neurosis. Make a primary positive diagnosis—only test if you have to.
3. Structure a program for them, for example, 'Mrs Jones, I have decided that we should meet for 15 minutes every second Wednesday at 10 am.'
4. During the consultation, demonstrate your genuine interest in the person's life, garden, work and so on; show less interest, even boredom, for the litany of complaints.

Other management guidelines include the following.

- Use reassurance with caution—it is insufficient by itself and should be soundly based.
- Be honest and maintain trust.
- Allow the patient a fair share of your time—this is your part of the contract. At the same time indicate that there are limits to your time (set rules).
- Be polite yet assertive.
- Avoid using labels of convenience and placebo therapy.
- Be honest about your understanding (or lack of understanding) of the problems.
- Remember that the consultation is often the therapy, without a prescription.
- Do not undermine other doctors. Avoid collusion.
- Have limited objectives—zealous attempts to cure may be inappropriate.
- Do not abandon the patient, however frustrating the relationship. Accept this as a legitimate role.
- Remain available if alternative therapies are sought by the patient.
- Take extra care with the 'familiar' patient and sometimes the patient who brings gifts. Maintain your professional role.

- If you are uncomfortable with counselling, consider early referral to a counsellor while maintaining contact in the future.
- You may have to accept that there are some people whom no one can help.

Complaints

Complaints from all groups of patients are common and disturbing. The main issues are usually a breakdown in communication or unmet expectations. This is likely to occur when something has gone wrong.

Tips to handle complaints include:⁷

- don't ignore them—deal with them as quickly as possible
- speak directly to the complaining patient, preferably face to face
- refer the complaint to the medical registration board or to your medical insurer for advice if there is a legal claim for negligence
- an appropriate strategy for other complaints is to acknowledge them in writing and follow up with a phone call
- recommend a meeting to discuss concerns
- check all facts surrounding the issue including detailed copies of reports and records of phone calls

An ABCDE of dealing with complaints is presented in TABLE 71.1 .

Table 71.1 The ABCDE of dealing with complaints

-
- | | |
|----------|---|
| A | Acknowledge the complaint. |
| B | Set Boundaries for the patient. |
| C | Show Compassion and Caring. |
| D | Determine the reason for the behaviour. |
| E | Escape or Exit, if there is an impasse. |
-

A ‘heartsink’ survival kit

A pilot workshop of managing ‘heartsink’ patients described by Mathers and Gask⁸ led to the formulation of a ‘heartsink survival’ model for the management of patients with somatic

symptoms of emotional distress.

The first part of the three-part model, which is called ‘feeling understood’, includes a full history of symptoms, exploration of psychosocial cues and health beliefs, and a brief, focused, physical examination. In the second stage, termed ‘broadening the agenda’, the basic aim is to involve discussion of both emotional and physical aspects during the consultation. It includes reframing the patient’s symptoms and complaints to provide insight into the link between physical, psychological and life events.

Page 852

In the third stage, ‘making the link’, simple patient education methods are used to explain the causation of somatic symptoms, such as the way in which stress, anxiety or depression can exaggerate symptoms. It also includes projection or identification techniques using other sufferers as examples.

The angry patient

Anger in patients and their relatives is a common reaction in the emotive area of sickness and healing. The anger, which may be concealed or overt, might be a combination of fear and insecurity. It is important to bear in mind that many apparently calm patients may be harbouring controlled anger. The practice of our healing art is highly emotive and can provoke feelings of frustration and anger in our patients, their friends and their relatives.

Anger is a normal and powerful emotion, common to every human being, yet with an enormous variety of expression. The many circumstances in medicine that provoke feelings of anger include:⁹

- disappointment at unmet expectations
- crisis situations, including grief
- any illness, especially an unexpected one
- the development of a fatal illness
- iatrogenic illness
- chronic illness, such as asthma
- financial transactions, such as high cost for services
- referral to colleagues, which is often perceived as failure
- poor service, such as long waits for an appointment
- problems with medical certificates
- poor response to treatment

- inappropriate doctor behaviour (e.g. brusqueness, sarcasm, moralistic comments, aloofness, superiority)

The patient's anger may manifest as a direct confrontation with the doctor or perhaps with the receptionist, with litigation or with public condemnation.

In an extreme example, a Melbourne doctor was shot and killed by an angry patient who had been denied a worker's compensation certificate for a claim considered unjustified.

When a patient expresses anger about the medical profession or our colleagues it may be directed at us personally and, conversely, if directed to us it may be displaced from someone else, such as a spouse, employer or other figure of authority.

What is anger?

Anger is a person's emotional response to provocation or to a threat to his or her equilibrium. If inappropriate, it is almost always the manifestation of a deeper fear and of hidden insecurity. Angry, abusive behaviour may be a veiled expression of frustration, fear, self-rejection or even guilt.

On the other hand, its expression may be a defence against the threat of feeling too close to the doctor, who could have an overfamiliar, patronising or overly friendly attitude towards the patient. Some patients cannot handle this threatening feeling.

Basically, anger may be a communication of fear and insecurity. The patient could be saying, 'I am afraid there is something seriously wrong with me. Are you doing everything to help me?'

Consulting strategies¹⁰

When one feels attacked unfairly, to react with anger is a natural human response. This response, however, must be avoided since it will damage the doctor–patient relationship and possibly aggravate the problem.

- The initial response should be to remain calm, keep still and establish eye contact.
- 'Step back' from the emotionally charged situation and try to analyse what is happening.
- Ask the patient to sit down and try to adopt a similar position (the mirroring strategy) without any aggressive pose.
- Address the patient (or relative) by the appropriate name, be it Mr or Mrs Jones or a first name.
- Appear calm, comfortable and controlled.
- Be interested and concerned about the patient and the problem.

- Use clear, firm, non-emotive language.
 - Listen intently.
 - Allow patients to ventilate their feelings and help to relieve their burdens.
 - Allow patients to ‘be themselves’.
 - Give appropriate reassurance (do not go overboard to appease the patient).
 - Avoid a judgmental approach
 - Allow time (at least 20 minutes).
- Page 853
- For the threatening aggressive patient, sit closest to the door to allow escape should the patient turn violent.

Analysing the responses

- Search for any ‘hidden agenda’.
- Recognise the relationship between anger and fear.

Recognising distress signals

It is important to recognise signs of deteriorating emotional distress:¹¹

- body language (demonstrative agitated movements or closing in)
- speech (either becoming quiet or more rapid and louder)
- colour (either becoming flushed or pale)
- facial expression (as above, tense, tightening of muscles of eye and mouth, loss of eye contact)
- manner (impatient, threatening)

Skilful consulting strategies should then be employed. It is worthwhile having a contingency plan, such as memorising a telephone number to summon security help.

Questions to uncover the true source of anger

The following represent some typical questions or responses that could be used during the interview.

Rapport building

- ‘I can appreciate how you feel.’
- ‘It concerns me that you feel so strongly about this.’
- ‘Tell me how I can make it easier for you.’

Confrontation

- ‘You seem very angry.’
- ‘It’s unlike you to be like this.’
- ‘I get the feeling that you are upset with ...’
- ‘What is it that’s upsetting you?’
- ‘What really makes you feel this way?’

Facilitation, clarification

- ‘I find it puzzling that you are angry with me.’
- ‘So you feel that ...’
- ‘You seem to be telling me ...’
- ‘If I understand you correctly ...’
- ‘Tell me more about this ...’
- ‘I would like you to enlarge on this point—it seems important.’

Searching

- ‘Do you have any special concerns about your health?’
- ‘Tell me about things at home.’
- ‘How are things at work?’
- ‘How are you sleeping?’
- ‘Do you have any special dreams?’
- ‘Do you relate to anyone who has a problem like yours?’
- ‘If there’s any one thing in your life that you would like to change, what would it be?’

Some important guidelines are summarised in TABLE 71.2 .

Table 71.2 Guidelines for handling the angry patient

Do	Don't
Listen	Touch the patient
Be calm	Meet anger with anger
Be comfortable	Reject the patient
Show interest and concern	Be a 'pushover'
Be conciliatory	Evasive the situation
Be genuine	Be overfamiliar
Allay any guilt	Talk too much
Be sincere	Be judgmental
Give time	Be patronising
Arrange follow-up	Be drawn into action
Act as a catalyst and guide	

The drug-dependent doctor shopper¹²

Nicholas Carr's five-step approach:

- 1. Elicit request for drug early:

'In what way did you think I could help?'

'What sort of thing did you have in mind?'

- 2. Respectful refusal and brief minimal explanation:

'I don't prescribe Valium (or requested drug).'

'Yes, I could but I choose not to.'

- 3. Avoid being drawn into the patient's agenda:

'I understand this doesn't suit you, but I don't prescribe these drugs.'

- 4. Depersonalise:

‘It’s nothing to do with you personally—this is the way I work.’

5. Offer alternative help:

‘I’m happy to talk about other ways of helping, but they don’t involve a prescription.’

Management

When confronted with an angry patient, the practitioner should be prepared to remain Page 854 calm, interested and concerned. It is important to listen intently and allow time for the patient to ventilate his or her feelings.

A skilful consultation should provide both doctor and patient with insight into the cause of the anger and result in a contract in which both parties agree to work in a therapeutic relationship. The objective should be to come to amicable terms which, of course, may not be possible, depending on the nature of the patient’s grievance.

If the problem cannot be resolved in the time available a further appointment should be made to continue the interview.

Sometimes it may be appropriate to advise the patient to seek another opinion. If the angry patient does have problems with relationships and seeks help, it would be appropriate to arrange counselling so that the patient acquires a more realistic self-image, thus leading to improved self-esteem and effectiveness in dealing with people. In addition, it should lead to the ability to withstand frustration and cope with the many vicissitudes of life—a most rewarding outcome for a consultation that began with confrontation.

Violence and dangerousness

Dangerousness has been defined as a ‘propensity to cause serious physical injury or lasting psychological harm to others’ and, in the context of people with mental illness, ‘the relative probability of their committing a violent crime’.¹³

Dangerousness is not related only to mental illness and, interestingly, most offenders have no psychiatric diagnosis. It is not an inherited, immutable characteristic of an individual but tends to surface on impulse in a particular context given a whole range of situational factors. Prediction of the risk of violence is not straightforward.

Various groups have been identified as contributing risk factors for violent conduct.¹³

- Schizophrenic psychoses, including: older male paranoid schizophrenics; younger males prone to act violently and impulsively, presumably due to hallucinatory commands
- Morbid jealousy: associated with delusions of infidelity
- Antisocial personality disorder

- Mood disorder: violence, usually associated with depression (rarely mania); parents with severe depression; history of suicide attempts in depression
- Episodic dyscontrol syndrome (similar to intermittent explosive disorder)
- Intellectual disability combined with personality disorder and behavioural disturbances
- Alcohol abuse or dependency
- Amphetamine or benzodiazepine abuse or dependency

From a management viewpoint, homicidal threats must be taken very seriously.

The diagnostic strategy model for antisocial behaviour in adults is presented in [TABLE 71.3](#).

Table 71.3 Antisocial behaviour in adults:
diagnostic strategy model

Probability diagnosis

- Functional (no medical or mental component)
- Drugs (alcohol, illicit or prescribed)
- Alcohol (acute or chronic)
- Antisocial personality disorder (esp. cluster B)
- Affective (mood) disorders
- Drug withdrawal (incl. alcohol, hypnotics)
- Mental impairment

Serious disorders not to be missed

Vascular:

- cerebrovascular disease (incl. SAH)
- acute coronary syndromes

Infections:

- encephalitis/meningitis
- HIV/AIDS
- septicaemia

Tumours:

- cerebral tumours

Other:

- post-ictal (epilepsy)
- delirium
- subdural haematoma

- psychosis (schizophrenia, bipolar, paraphrenia)
 - schizotypal personality disorder
-

Pitfalls (often missed)

Head injury

Fluid and electrolyte imbalance

Dementia (esp. early)

Rarities:

- neurosyphilis
 - prion disease (e.g. CJD)
 - premenstrual dysphoria syndrome
-

Seven masquerades checklist

Depression (major)

Diabetes (hypoglycaemia)

Drugs (iatrogenic/social, illicit)

Thyroid/other endocrine (hyper/hypothyroid)

Is the patient trying to tell me something?

Consider conversion disorder (hysterical fugue)

Malingering/fabrication

Severe anxiety/panic

Suicide and parasuicide

The haunting issue of suicide and parasuicide is presented in [CHAPTER 10](#) . The Page 855 disturbed patient is always a suicide risk rather than a homicide risk. The importance of recognising depression with an associated suicide risk in the elderly patient has been emphasised heavily in [CHAPTER 69](#) .

Facts and figures¹⁴

- More than 90% of suicides occur without underlying chronic conditions but most people are significantly depressed at the time.
- In Australia, suicide is the second most common cause of death between the ages of 11 and 25 years. Children as young as 5 years of age have committed suicide.
- Those who talk about suicide may attempt it later.
- About half of those committing suicide have seen a doctor within their last month of life.

- Around 80–90% of suicides have given clear or subtle warnings to family, friends or doctors.
- There is no evidence that asking patients about suicidal ideation provokes suicidal acts.
- Doctors in Australia and other Western countries have a high suicide rate.

Suicide risk

Blumenthal's¹⁵ overlapping model lists five groups of risk factors (see FIG. 71.1):

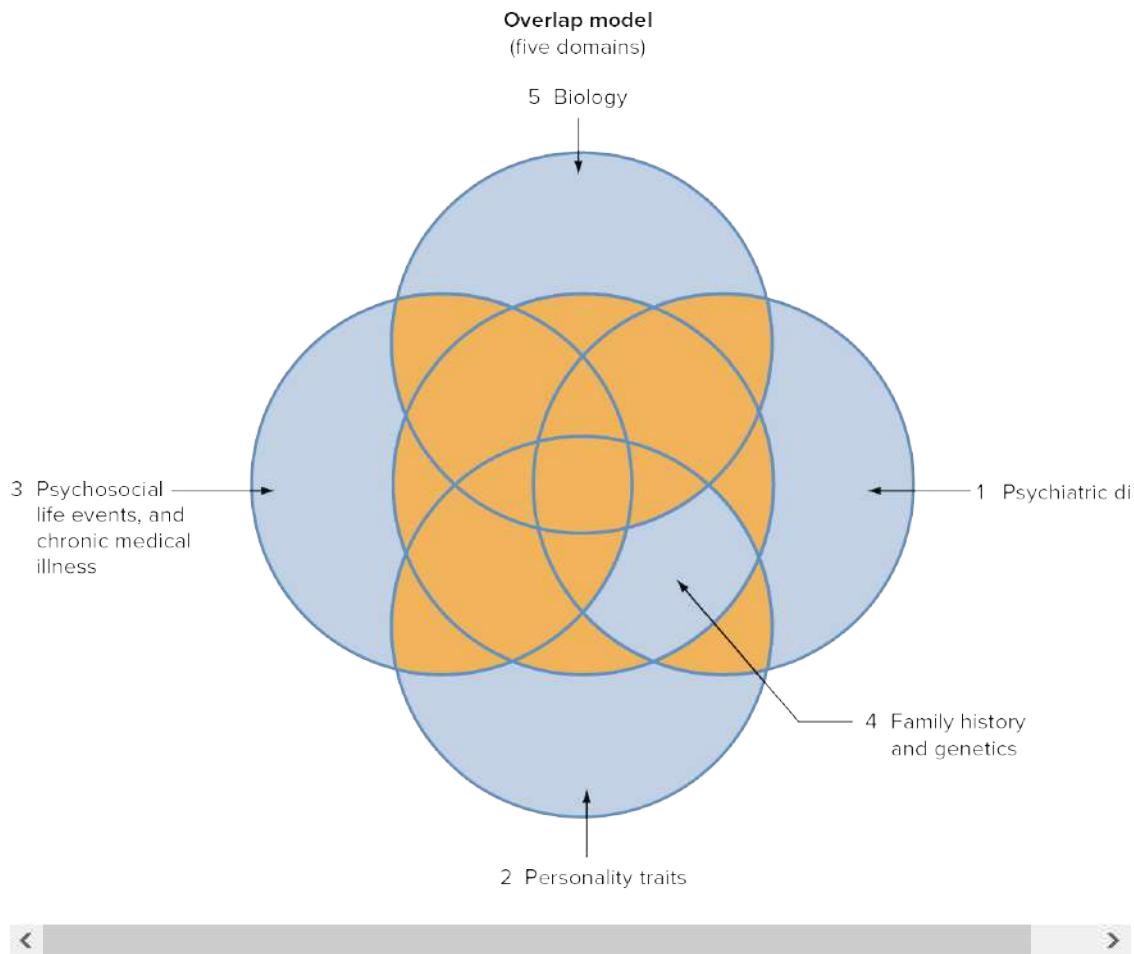


FIGURE 71.1 Overlap model for understanding suicidal behaviour¹⁶

I. Psychiatric disorders:

- affective disorder and alcohol abuse in adults
- schizophrenia
- depression and conduct disorder in young people

- anorexia nervosa
-). Personality traits:
 - impulsiveness and aggression
-). Environmental and psychosocial factors:
 - poor social supports
 - chronic medical illness (e.g. AIDS)
 - significant loss
-). Family history and genetics (both nature and nurture):
 - emulation of relatives
 - specific ethnic groups in custody
-). Biological factors:
 - possible serotonin deficiency

Parasuicide

Parasuicide is attempted suicide; in many cases, patients are drawing attention to themselves as a ‘plea for help’. The patient presenting with threatened suicide or self-harm, be it a traumatic wound or overdosage of medication or a toxic agent, is a ‘heartsink’ challenge. Consider borderline personality disorder, especially in the adolescent or young adult. It is important for the GP to take an active role in the support of the patient and family after discharge from hospital, but preferably in conjunction with a psychiatric or counselling service. Arrange frequent consultations at first and ensure adequate follow-up, especially for missed appointments.

Page 856

Personality disorders

People with personality disorders may become very distressed and acutely disturbed under stress or provocation, and this may involve dramatic scenes, including public suicide threats. It is important to recognise personality disorders because they usually cause considerable distress to the patients, their family, society and GPs. The prevalence is estimated as 11–12%.

In practice, the personality disorders of most concern are those that present with hostility, either verbal or physical, particularly if a suicide or homicide threat is involved. It is a mistake to assume that those patients who manifest violent or psychopathic behaviour have a personality disorder or, conversely, that the meek and mild are free from personality disorder.

The diagnosis of personality disorder can be difficult. As practitioners we tend to have a ‘gut feeling’ about the diagnosis but often find it difficult to classify the personality of the patient and then to manage it appropriately.

The main characteristics of a personality disorder are:¹⁷

- lack of confidence and low self-esteem
- long history from childhood
- difficulties with interpersonal relationships and society
- recurrent maladaptive behaviour
- relatively fixed, inflexible and stylised reaction to stress
- minimal insight
- perception of difficulties as external to themselves

The medical/psychiatric significance:

- maladaptive relationships with GPs and society
- problem of sexually dysfunctional lives
- risk of substance abuse and self-destructive behaviour
- prone to depression and anxiety (usually low grade)
- susceptible to ‘breakdown’ under stress

Personality is the result of a genetic template and the continuing interaction of the person with outside influences (peer pressures, family interactions, influential events) and personal drives in seeking an identity. The outcome is a unique behaviour pattern manifesting as a personality trait or character reflective of the individual’s self-image and fundamental to his or her sense of personal identity.¹⁸

Although personality is unique, it is possible to make a hypothesis that one is normal or abnormal. If abnormal, it is possible to stereotype it according to the predominant symptoms or behaviours.

Using the International Classification of Disease (ICD-10) and the DSM-5 classification, various subtypes are readily identifiable (see TABLE 71.4),¹⁹ which can be considered in three main groups. There is a considerable overlap between the subtypes within a group²⁰ and it is more important to understand the specific features of a person’s personality than to categorise them.²¹

Table 71.4 Summary of main personality disorders—based on DSM-5

Main cluster group	Subtypes	Main features of disorder
Cluster A Withdrawn <i>Synonyms:</i> <ul style="list-style-type: none">• odd• eccentric	Paranoid Schizoid Schizotypal	Suspicious, oversensitive, argumentative, defensive, unforgiving, hyperalert, cold and humourless Shy, emotionally cold, introverted, detached, avoids close relationships, indifferent to praise and criticism Odd and eccentric, sensitive, suspicious and superstitious, socially isolated, odd speech, thinking and behaviour. Falls short of criteria for schizophrenia
Cluster B Antisocial <i>Synonyms:</i> <ul style="list-style-type: none">• dramatic• emotional• sociopathic• flamboyant• erratic	Antisocial (sociopathic, psychopathic) Histrionic (hysterical) Narcissistic ('prima donna') Borderline ('impulsive')	Impulsive, insensitive, selfish, callous, superficial charm, lack of guilt, low frustration level, doesn't learn from experience, deceitful, relationship problems (e.g. promiscuous), reckless disregard for safety of self and others, lack of self-control Self-dramatic, egocentric, immature, vain, dependent, theatrical, manipulative, easily bored, emotional scenes, inconsiderate, seductive, craves attention and excitement Morbid self-admiration, grandiose, exhibitionist, insensitive, craves and demands attention, exploits others, preoccupied with power, success, beauty, lacks interest in and empathy with others, bullying, arrogant, insightless, 'charming, disarming, alarming', prone to fits of destructive anger and revenge, sense of entitlement Confused self-image/identity, impulsive, reckless, emptiness, 'all or nothing' relationships—emotionally unstable and intense,

damaging reckless behaviour, full of inappropriate anger and guilt, fear of abandonment, lacks self-control, uncontrolled gambling, spending, etc.

Note: High incidence of suicide and parasuicide; drug abuse

Cluster C Dependent <i>Synonyms:</i> <ul style="list-style-type: none">• anxious• fearful• inhibited	Avoidant (anxious) Dependent Obsessional (obsessive–compulsive, anankastic)	Anxious, self-conscious, fears rejection, timid and cautious, low self-esteem, overreacts to rejection and failure Passive, weak-willed, lacks vigour, lacks self-reliance and confidence, overaccepting, avoids responsibility, seeks support, excessive need to be cared for Rigid, perfectionist, pedantic, indecisive, egocentric, preoccupied with orderliness and control, cautious
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Note: The 'dark antisocial triad': narcissism, psychopathy (seeks control) and Machiavellianism (the end justifies the means).²²

Practice tip

The cardinal feature of antisocial personality disorder is lack of empathy.

The antisocial personality disorders (ASPD) group (1–2% of population) tend to come to the attention of GPs more frequently, with some individuals representing 'heartsink' patients because of demanding, angry or aggressive behaviour. They are more common among people in prison. The withdrawn group are typically distant, suspicious and socially isolated but fall short of a true psychotic syndrome. GPs have problems communicating with them because they are often suspicious, which can make proper physical examination and management difficult.

In the dependent and inhibited groups, which may overlap with an anxiety state, the main features are nervousness, timidity, emotional dependence and fear of criticism and rejection. They are frequent attenders (the 'fat file' syndrome) and are often accompanied by friends and relatives because of their insecurity. It is imperative to think laterally and of borderline personality disorder in the person, especially young, presenting with suicide attempts, an eating disorder or uncontrolled behaviour.

Management

The best treatment is a supportive, ‘therapeutic’ community and an understanding and supportive GP. Psychotherapies are the key to long-term treatment. It is vital to understand that people with personality disorders perceive the world from a fundamentally different perspective. Problematic patients, if agreeable, may respond well to psychological intervention and behavioural techniques, especially operant conditioning (reinforcing acceptable behaviour) and aversive conditioning (correcting inappropriate behaviour).^{16,17} CBT has the most to offer. These therapies are best administered by clinicians with specialised training and expertise.¹⁶

The borderline and narcissistic disorders in particular respond well to specific types of psychotherapeutic intervention. Patients’ self-esteem needs careful support while maladaptive modes of behaviour are confronted. Hospitalisation is rarely required except for those at risk of suicide (e.g. antisocial patients).

Medication has limitations but may be useful to treat those individuals who temporarily decompensate into a psychosis, an anxiety state or depression. One study has shown that antipsychotic medication in low dosage (e.g. haloperidol 5 mg daily) is effective in treating the problematic behaviours in paranoid and some antisocial personality disorders.²³

There are dangers to the therapist and it is important not to ‘buy into’ a particular psychopathy, especially with seductive, manipulative or paranoid patients.¹⁵

Patient education resources

Hand-out sheets from *Murtagh’s Patient Education* 8th edition:

[Page 858](#)

- Anger management
- Borderline personality disorder
- Personality disorders
- Schizophrenia

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Part 5 Chronic disease management

Page 860

72 Allergic disorders including hay fever

The nostril membrane is so irritable that light dust, contradiction, an absurd remark—anything—sets me sneezing and I can be heard in Taunton with a favourable wind, a distance of six miles. Turn your mind to this little curse. If consumption is too powerful for physicians, at least they should not suffer themselves to be outwitted by such little upstart disorders as the hay fever.

SYDNEY SMITH, LETTER TO DR HOLLAND, 1835

Allergic disorders affect approximately 20% of the population. The clinical spectrum of allergic disorders includes asthma, rhinitis, atopic dermatitis, drug allergy, food allergy, insect sting allergy and anaphylaxis.¹ The most common allergies are those associated with IgE-mediated (immediate or type 1) hypersensitivity, such as allergic rhinoconjunctivitis (hay fever), atopic dermatitis (eczema) and allergic asthma.²

Less commonly encountered but of increasing clinical importance in the community are IgE-mediated allergies to foods such as peanuts and/or other nuts and seafoods (crustaceans or molluscs), which may cause urticaria, angioedema, anaphylaxis and even death. Peanuts are one of the most common causes of food-induced anaphylaxis in adults. A clinically significant cross-reactivity between peanuts and other legumes is uncommon, but allergy to tree nuts such as almonds and walnuts may occur in up to 50% of those with peanut allergy.¹ Another special case is the oral allergy syndrome, in which people with some degree of seasonal allergy to grass pollens or birch pollen suffer oral itch and swelling when they come into contact with certain fruits. This problem may be alleviated by desensitisation to pollens.¹

Natural rubber latex allergy is an increasingly important cause of type 1 hypersensitivity, affecting particularly medical and paramedical personnel, and patients who have had multiple operations or procedures. Diagnosis is suggested by history and confirmed by specific skin tests or the detection of serum-specific IgE. The development of urticaria on contact with latex is highly suggestive of underlying type 1 hypersensitivity. An interesting association between latex allergy and sensitivity to fruit occurs most commonly with banana, kiwifruit or avocado.¹

Atopy

Atopy occurs in those 45% of the population who have an inherited tendency for an exaggerated IgE antibody response to common environmental antigens.² It is not a disease in itself. There will be a positive response to one or more allergen skin-prick tests and usually a family history of allergic disorders. Of those who are atopic, one-half to one-third manifest an allergic disorder, most commonly allergic rhinitis, asthma, atopic dermatitis or allergic gastroenteropathy.

Common allergens causing immediate hypersensitivity

It is helpful to consider important allergen exposure during history taking. TABLE 72.1 lists sources of common allergens.

Table 72.1 Sources of common allergens^{1,2}

Inhalants

Pollens, domestic animals, house dust mites, mould spores, cockroaches

Foods

Peanuts, fish, shellfish, milk, eggs, wheat

Other

Drugs, latex, insect venoms, occupational

Inhalant allergies

Allergic rhinoconjunctivitis and asthma are the main manifestations. The history provides a strong pointer to the causative allergen. If symptoms are seasonal, pollen allergy is most likely; perennial symptoms may indicate an allergy to dust mites, household pets or moulds. Certain activities that precipitate symptoms may also provide a clue—these include mowing lawns, dusting and vacuuming.

Food allergy and intolerance³

Food allergy usually manifests in infancy and childhood, with symptoms ranging from severe urticarial-type reactions to gastrointestinal symptoms such as anorexia, nausea, vomiting and spitting up of food, colic, diarrhoea and failure to thrive.

Page 861

Commonly implicated foods include milk and other dairy products, eggs and peanuts. Other

foods include oranges, soy beans, nuts, chocolate, fish, shellfish and wheat.

The allergic reactions are not to be confused with non-immunological food intolerances such as lactose intolerance.

A food intolerance is an adverse reaction to a specific food or food ingredient. It is regarded as a food allergy if the reaction is immune based. Food allergies can be simply classified as:

- immediate reactions—occurring within 2 hours
- delayed reactions—occur up to 24 hours after ingestion

IgE-mediated food reactions³

These are immediate immune-mediated responses to a foreign glycoprotein which are relatively easy to diagnose.

Clinical features

- Typically in infants and toddlers
- Usually occur within 30–60 minutes
- Due to release of mast cell mediators
- Produce flushing or blotchiness/pallor (if severe)
- Itchy oropharynx
- Itchy, runny nose and eyes
- Wheeze
- Dizziness and confusion
- Urticaria—facial or generalised (see FIG. 72.1)



FIGURE 72.1 Acute urticaria in a child caused by a sensitivity to aspirin

- Feeling of intense fear (angor animi)
- Angioedema of face and airway

- Vomiting, diarrhoea and abdominal colic (immediate or soon after)
- ± Anaphylaxis with wheezing, etc.
- Death can occur (especially if asthma history)
- Big three foods—cow's milk, egg, peanuts
- Also: soy beans; fish, especially shellfish; wheat; tree nuts; various fruits and vegetables
- Cow's milk can cross-react with goat's milk and soy protein
- Frequently resolves by 3–5 years
- In adults the foods are mainly peanuts, tree nuts, fish and shellfish

Management

- Document diet, symptoms, past history, family history
- Refer for specialist allergy advice
- Provide patient education sheet
- Advise avoidance of suspected food
- IgE reactions investigated with skin testing
- Consider provision of an adrenaline autoinjector

Page 862

Non-IgE-mediated food reactions³

These are less common and are usually delayed, occurring within 24–48 hours of food ingestion—but some reactions may be immediate. The real explanation is not clear. This includes cow's milk protein intolerance with both breast milk- and formula-fed infants. It affects 2% of infants under 2 years with most resolving by 2–3 years of age.

Clinical features

- Gastrointestinal symptoms (e.g. vomiting, diarrhoea, abdominal colic)
- May be malabsorption, weight loss, failure to thrive (rare)
- Aggravation of atopic dermatitis
- Severe reactions possible
- Main foods—cow's milk, soy proteins

- Uncommon after 3 years of age

Management

- Elimination of suspected food, then formal food challenge
- For milk protein intolerance first-line treatment is a formula containing cow's milk protein hydrolysate. Don't use soy-based formulas under 6 months since many are also soy protein intolerant.
- Skin testing usually not helpful and may be risky

Food protein-induced enterocolitis syndrome (FPIES)

This is seen in young infants usually <6 months and is usually due to cow's milk, soy or cereals. It can be seen in breastfed infants and older children. A typical reaction is delayed onset of projectile vomiting and protracted diarrhoea. The stool contains blood and eosinophils. Treatment is with substance elimination.

Food intolerances⁴

In general practice it is common to see a variety of food intolerances that are not immune (allergic) or psychologically based. The food constituents represent an important group. The intolerances can be grouped as:

- fructose intolerance from excessive ingestion of fruit juices and soft drinks
 - lactase deficiency from milk
 - histamine-related reactions from strawberries, tomatoes
 - chemical triggers:
 - aspirin, tartrazine, sodium metabisulphite—triggering rhinitis and asthma
 - aspirin, tartrazine, benzoic acid—triggering chronic urticaria
 - others—salicylates (manufactured or natural), amines, preservatives and colourings
- Note:*
- foods with high-content natural salicylate include: dried fruits, pineapple, apricots, oranges, cucumbers, grapes, honey, olives, tomato sauce, wines, tea, herbs
 - foods likely to contain tartrazine (food colouring) include: bottled sauces, cakes (from shops), coloured fizzy drinks, fruit cordial, custard, coloured sweets, ice-cream and lollies, jam

Symptoms

- Irritability, behavioural problems
- Gastrointestinal (e.g. infant colic, diarrhoea, irritable bowel syndrome)
- Respiratory—rhinitis, asthma
- Headache/migraine

Management

- Document diet, reactions, past/family history
- Elimination diet and controlled food challenge
- Referral for specialist allergy advice
- Radioallergosorbent (RAST) testing is indicated where skin testing contraindicated

Peanut allergy

Peanuts are one of the most common causes of food-induced allergy including anaphylaxis in adults. The diagnosis is confirmed by demonstration of peanut-specific IgE by either skin-prick tests with peanut extract or RAST testing. Reaction to peanuts usually begins within minutes of ingestion, the first symptoms being oropharyngeal itching or burning. Flushing, urticaria, wheeze, stridor, angioedema and collapse may follow.⁵ The combination of peanut allergy and asthma is dangerous, as evidenced by fatal or near-fatal reactions in young children.⁶ The key to management is avoidance of peanut-containing foods. Desensitisation is currently not recommended. Those at risk should carry an anaphylaxis kit (see TABLE 72.2).

Table 72.2 Adult anaphylaxis kit²

Autoinjector 300 mcg adrenaline 1:1000 IM injection

Inject into outer thigh muscle at first sign of swelling of throat or tongue, or other reaction (e.g. breathlessness)

Medihaler-Epi MDI (adrenaline metered aerosol spray)

Spray 10–20 times in milder reactions only (e.g. local lip tingling or swelling)

Oral antihistamines

e.g. 10 mg loratadine tablets (× 2)
Take 1 tablet after adrenaline injection

Prednisolone 25 mg tablets (× 2)

Take immediately after adrenaline

Doses for children: 15–30 kg—150 mcg; 30 kg—300 mcg

Somewhat counterintuitively, recent studies have entirely reversed previous advice around exposure to peanuts in infancy. The Australian Society of Clinical Immunology and Allergy (ASCIA) recommends the early introduction of peanut butter/paste to infants, before 12 months. The greatest benefit is actually to infants at higher risk; infants with severe eczema and/or egg allergy can reduce their risk of developing peanut allergy by around 80%.⁷

Risk-minimisation strategies for food anaphylaxis¹

- Achieve optimal asthma control (unstable asthma is the major risk factor)
- Notify others: school, homes, restaurants
- Carry treatment: action plan and adrenaline
- ‘Touch test’ food on external lip before consumption
- Avoid skin preparations containing natural foods

Page 863

Egg allergy

Previous advice was to avoid introducing potentially allergenic foods to babies. The evidence now shows that babies who are given foods such as peanuts, eggs and seafoods in the first year of life develop fewer allergies than babies where these foods are strictly avoided. The Australian Society of Clinical Immunology and Allergy (ASCIA) has detailed information about introducing foods on its website.⁸

Current vaccinations do not include egg; it is present in only minute amounts in the MMR vaccine.

Latex allergy

The clinical manifestations of type 1 hypersensitivity reactions to latex protein are wide-ranging, from urticaria to life-threatening anaphylaxis and death. It is believed that some episodes of intra-operative anaphylaxis are due to the patient—who has been sensitised to latex—reacting after mucosal contact with gloves worn by operating staff. Many institutions now provide latex-free operating suites in response to this serious problem.⁹

Latex allergy is thus a significant problem for at-risk people, including health care workers, patients with spina bifida or other spinal cord abnormalities, and those who have had multiple operations. The greatest risk is contact with ‘dipped’ rubber products (e.g. gloves, condoms, balloons). Some hard rubber products may not pose a risk.

Symptoms

- Contact dermatitis (type 4 hypersensitivity), urticaria, worsening atopic dermatitis, allergic rhinoconjunctivitis, asthma, allergy to multiple fruits and possibly anaphylaxis

Diagnosis

Skin-prick tests (dangerous and best left to experts) are more sensitive than blood tests at this stage but carry a risk of anaphylaxis. Measurement of serum-specific IgE is safe although less sensitive. Contact allergy (type 4) is identified by patch testing.

Management

Health care workers who are allergic can never again wear latex gloves.¹⁰

Tests for specific IgE

Skin-prick tests

This is the preferred method as results can be read at the first consultation, provided high-quality allergen preparations are used. A positive test alone may be of no diagnostic significance if the patient is asymptomatic to the specific allergens. A negative test is very useful for excluding IgE-mediated allergy.

Detection of serum-specific IgE

A number of tests, including RAST tests¹ and ELISA tests, measure allergen-specific IgE in the serum. They are no more accurate than skin testing, are expensive and do not provide an immediate result.

Indications include: history and skin tests not matching, extensive eczema, dermographism, infants and very young children, immunotherapy work-up, antihistamine use in past 48 hours.

Management principles¹

Allergen avoidance

If relevant from history and skin-prick testing, special attention should be paid to reducing exposure to house dust mites and mould, to pet selection and specific food avoidance. Change of occupation and environment may be necessary for some people.

Pharmacotherapy

Drugs are used to alleviate symptoms where avoidance methods have failed or are impractical. Examples include antihistamines (H_1 -receptor and H_2 -receptor antagonists), adrenaline (emergency use), sodium cromoglycate, corticosteroids, some anticholinergics and sympathomimetics.

Immunotherapy (desensitisation)

This involves repeated administration of small, increasing doses of allergen by subcutaneous injection. This is the treatment of choice for severe wasp or bee venom allergy and for resistant allergic rhinoconjunctivitis where a single causative allergen can be identified. Patients should be observed for at least 45 minutes after each injection and adequate resuscitation facilities are essential.

Management of specific allergic disorders

- Asthma—see [CHAPTER 73](#)
- Atopic dermatitis—see [CHAPTER 113](#)
- Urticaria—see [CHAPTER 112](#)
- Anaphylaxis and angioedema—see [CHAPTER 120](#)

[Page 864](#)

Rhinitis

Refer to [CHAPTER 48](#).

The classification of rhinitis can be summarised as:

- seasonal allergic rhinoconjunctivitis = hay fever
- perennial rhinitis:

allergic (often due to house dust mites)

non-allergic = vasomotor: eosinophilic, non-eosinophilic

Allergic rhinitis^{10,11}

Definition

Allergic rhinitis May be seasonal or perennial. It can be classified as either intermittent (lasting for <4 days of the week or <4 weeks) or persistent (lasting >4 days of the week or >4 weeks).

The severity of symptoms is classified as either mild (normal function including sleep and only slightly troublesome symptoms) or moderate/severe (troublesome symptoms with impairment of activities).¹⁰ Its lifetime prevalence has increased worldwide, affecting 20% of the adult population and up to 40% in children; 60% have a family history. It varies from 5–20% with a peak prevalence in children and young adults up to 20%.¹² The symptoms are caused by release of powerful chemical mediators such as histamine, serotonin, prostaglandins and leukotrienes from sensitised mast cells.¹²

Seasonal allergic rhinoconjunctivitis (hay fever)

This is the most common type of allergic rhinitis and is due to a specific allergic reaction of the nasal mucosa, principally to pollens. The allergens responsible for perennial allergic rhinitis include inhaled dust, dust mite, animal dander and fungal spores.

Most cases of hay fever begin in childhood with one-half of eventual cases having the problem by the age of 15 and 90% by the age of 30.¹³ Approximately 20% suffer from asthma.

While those with hay fever tend to have widespread itching (nose, throat and eyes), those with perennial rhinitis rarely have eye or throat symptoms but mainly sneezing and watery rhinorrhoea. Nasal polyps are associated with this disorder (refer to [CHAPTER 48](#)).

Management

Management consists of four main areas:

1. appropriate explanation and reassurance
2. allergen avoidance
3. pharmacological treatment
4. immunotherapy

Intranasal corticosteroids which reduce inflammation and nasal secretions are first-line treatment for moderate to severe cases.¹⁴

Advice to patients

- Keep healthy, eat a well-balanced diet, avoid ‘junk food’ and live sensibly with balanced exercise, rest and recreation. If your eyes give you problems, try not to rub them, avoid contact lenses and wear sunglasses.
- Avoid using decongestant nose drops and sprays: although they soothe at first, a worse effect occurs on the rebound.

- Avoidance therapy: avoid the allergen, if you know what it is (consider pets, feather pillows and eiderdowns).
- Sources of the house dust mite include bedding, upholstered furniture, fluffy toys and carpets. Seek advice about significantly reducing the dust in your bedroom or home, especially if you have perennial rhinitis.
- Pets, especially cats, should be kept outside.
- Avoid chemical irritants such as aspirin, smoke, cosmetics, paints and sprays.

Allergen avoidance

This is difficult during the spring pollen season, particularly where patients are living in high-pollen (e.g. country farming) areas, or spending considerable time outdoors in the course of work or sporting and recreational activities.

Treatment (pharmacological)¹⁰

Therapy can be chosen from:

1. antihistamines:

- oral (not so effective for vasomotor rhinitis)
- intranasal spray (rapid action)
- ophthalmic drops

2. decongestants (oral or topical)

3. sodium cromoglycate

Page 865

- intranasal: powder insufflation or spray

- ophthalmic drops for associated conjunctivitis

4. corticosteroids

- intranasal (not so effective for non-eosinophilic vasomotor rhinitis)
- oral (very effective if other methods fail)
- ophthalmic drops for allergic conjunctivitis

Immunotherapy

Consider hyposensitisation/immunotherapy when specific allergens are known (very important)

and conventional response is inadequate. Immunotherapy to grass pollen is generally very effective and should be considered in moderate to severe springtime hay fever. Immunotherapy by injection or oral administration can be labour-intensive, often taking years.

Antihistamines

Oral antihistamines are the first line of treatment for seasonal hay fever and are generally effective where symptoms are intermittent, or when used prophylactically before periods of high pollen exposure. The newer 'non-sedating' antihistamines that do not cross the blood–brain barrier are used in preference to the first-generation drugs, although some degree of sedation may occur even with these. A list of non-sedating antihistamines is presented in TABLE 72.3. It is claimed by some that the newer topical preparations (levocabastine and azelastine), as intranasal sprays, are rapidly effective for an exacerbation of symptoms. If sedation is desirable (e.g. overnight), a sedating antihistamine can be used.

Table 72.3 Non-sedating antihistamines (oral regimens)

Generic name	Onset	Adult dosage
Cetirizine	Rapid	10 mg daily
Desloratadine	Very rapid	5 mg daily
Fexofenadine	Rapid	60 mg bd
Loratadine	Very rapid	10 mg daily

Oral decongestants

Oral sympathomimetics, either used alone or in combination with antihistamines (where they may help reduce drowsiness), may be of some value, particularly where nasal discharge and stuffiness are major symptoms. Side effects include nervousness and insomnia. They should be used cautiously in patients with hypertension, heart disease, hyperthyroidism, glaucoma and prostatic hypertrophy.

Examples:

pseudoephedrine HCl 60 mg (o) tds (max. 240 mg/day), or 120 mg controlled release (o) bd

Intranasal therapy^{10,11}

Intranasal decongestants should be used for limited periods only (i.e. less than a week) or intermittently (3–4 doses per week) because of the potential problems with rebound congestion and rhinitis medicamentosa. They are often of particular value during the first week of treatment

with intranasal corticosteroids (where the onset of action is delayed several days), improving nasal patency and allowing more complete insufflation of the corticosteroids. Adverse reactions similar to those of oral decongestants may occur.

Intranasal sodium cromoglycate acts by preventing mast cell degranulation and is effective without serious side effects. The capsule variety must be used (the spray form requires 1–2 hourly dosage to be effective); it is useful in perennial allergic rhinitis but is not as effective as intranasal corticosteroids for springtime hay fever.

Intranasal corticosteroid sprays are the most effective agents for treating seasonal allergic rhinitis. Side effects are minimal and adrenal suppression is not a problem with normal usage. Patients should be informed that these medications will not give immediate relief (often taking 10–14 days to have peak effect) and must be used continuously throughout the hay fever season for at least 6–8 weeks. Local side effects include dryness and mild epistaxis.

Intranasal antihistamines group—includes azelastine and levocabastine—are effective at relieving itching and sneezing.

TABLE 72.4 lists intranasal preparations for rhinitis.

Table 72.4 Intranasal preparations for rhinitis

	Brand name	Dosage	Comments
Sodium cromoglycate	Rynacrom powder (capsules)	Insufflate 1 capsule, qid 2%	Compliance a problem
	Rynacrom nasal spray	Spray 4–6 times daily 4%	
		Spray 2–4 times daily	
Beclomethasone dipropionate 50 mcg/spray	Beconase Hayfever	100 mcg spray each nostril bd or tds	
Budesonide 64 mcg/spray	Budamax nasal	1–2 sprays each nostril daily	
	Rhinocort nasal		
Ciclesonide 50 mcg/spray	Alvesco, Omnaris	2 sprays each nostril daily	

Fluticasone furoate 27.5 mcg/spray	Avamys	2 sprays each nostril daily, reducing to 1 spray	
Fluticasone propionate 50 mcg/spray	Beconase Allergy 24 hour aqueous	2 sprays each nostril daily, reducing to 1 spray	
Mometasone furoate 50 mcg/spray	Nasonex	2 sprays per nostril daily	
Triamcinolone 55 mcg/spray	Telnase	2 sprays each nostril daily, reducing to 1 spray	
Ipratropium bromide	Atrovent	1–2 sprays per nostril tds prn	Useful for vasomotor rhinitis and profuse rhinorrhoea Care needed with elderly
Azelastine	Azep	1 spray each nostril bd	Antihistamine
Levocabastine 0.05%	Livostin	2 sprays each nostril bd	Antihistamine, max. 8 weeks
Various sympathomimetics (e.g. phenylephrine)		2, 3 or 4 times daily (max. 7 days)	Short-term use only Care with elderly, prostatic hypertrophy

Ophthalmic preparations

Sodium cromoglycate eyedrops are usually very effective for springtime conjunctivitis. They can be used as necessary (no dosage limit) and are most helpful when used prophylactically before periods of high pollen exposure. Decongestant eyedrops may also be helpful (care with narrow angle glaucoma), while corticosteroid eyedrops are reserved for resistant allergic conjunctivitis and should be used with care to exclude infection and glaucoma. Antihistamine eyedrops antazoline and levocabastine are yet another option.

Other treatments

Corticosteroids (oral)

These can be very effective where other treatments or methods have failed. A 6–10-day short course can be used. An example of a 6-day ‘rescue course’ is prednisolone 25, 25, 20, 15, 10, 5 mg daily doses.

Page 866

Ipratropium bromide (Atrovent)¹⁵

The nasal preparation of this topical anticholinergic is often very effective when rhinorrhoea is the major problem.

Leukotriene receptor antagonist

Regarded as equivalent to oral antihistamines, they have a place in the management of children with concurrent asthma and hay fever (e.g. montelukast).

Surgery

Inferior turbinate reduction aims to reduce the size of turbinates and so reduce nasal obstruction when congested.

Guidelines from elite task force¹⁶

1. For initial treatment of seasonal allergic rhinitis in people aged 12 or older, routinely prescribe monotherapy with an intranasal corticosteroid rather than an intranasal corticosteroid in combination with an oral antihistamine.
2. For initial treatment of seasonal allergic rhinitis in those aged 15 or more, recommend an intranasal corticosteroid over a leukotriene receptor antagonist.
3. For treatment of moderate to severe seasonal allergic rhinitis in people 12 or older, you may recommend the combination of an intranasal corticosteroid and an intranasal antihistamine initially.

TABLE 72.5 summarises recommended steps in management.

Table 72.5 Summary of recommended treatment steps for allergic rhinitis¹⁰

Patient education

Allergen avoidance (if possible)

Mild cases:

- less-sedating? antihistamines including levocabastine nasal spray ±
- decongestant (e.g. pseudoephedrine)

Moderate to severe (persistent):

- intranasal corticosteroids (as preventer—the most effective)
 - sodium cromoglycate (Opticrom) eyedrops
 - oral corticosteroids (if topicals ineffective)
 - immunotherapy if applicable
-

When to refer

- Where surgical intervention is required, such as with nasal obstruction from polyps, bulky nasal turbinates and deviated septum
- For immunotherapy

Page 867

Practice tips

- Avoid long-term use of topical decongestant nasal drops.
- Avoid topical antihistamine preparations.
- Prescribe sodium cromoglycate eyedrops for the hay fever patient with itchy eyes.
- Be careful of severe systemic reactions that can occur with intradermal skin testing and with immunotherapy. Resuscitation facilities should be available.

Resource

Australasian Society of Clinical Immunology and Allergy: www.allergy.org.au

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73 Asthma

After I'd written to you yesterday I had an attack of asthma and an incessantly running nose, which forced me to tramp about, lighting cigarettes at every tobacconist etc. And worse was to come: I went to bed about midnight, feeling all right after spending a long time inhaling smoke, but 3 or 4 hours later came the real attack of the summer.

MARCEL PROUST, LETTER TO HIS MOTHER, 1901

Asthma, which is an inflammatory disorder, is defined by the presence of both of the following:¹

- excessive variation in lung function ('variable airflow limitation', i.e. variation in expiratory airflow that is greater than that seen in healthy people)
- respiratory symptoms (e.g. wheeze, shortness of breath, cough, chest tightness) that vary over time and may be present or absent at any point in time

In young children in whom lung function testing is not feasible, including most preschool children, asthma is defined by the presence of variable respiratory symptoms.

Key facts and checkpoints

- Asthma continues to be underdiagnosed and undertreated.² It is increasing worldwide.
- It has an unacceptable mortality rate: 421 deaths in Australia in 2019.³
- One child in nine (age 0–14 years) reports having asthma (usually in a mild form).⁴
- It tends to develop between the ages of 2 and 7 years, but can develop at any age.
- Most children present with a cough.
- Most children are free from it by puberty.

- About one adult in eight has or has had asthma.
- The focus of management should be on prevention; an acute asthmatic attack represents failed treatment.
- Measurement of function is vital as 'objective measurement is superior to subjective measurement'.
- Spirometry is the key investigation.
- Inhaled corticosteroids are the cornerstone of asthma treatment.
- Medicines should be prescribed at the lowest strength that works. Patients should not be left on combination or high-dose inhalers without regular review.
- Avoid concomitant medication that may exacerbate asthma (e.g. beta blockers, aspirin, NSAIDs).

Pathophysiology¹

Chronic asthma is an inflammatory disease with the following pathological characteristics:

- infiltration of the mucosa with inflammatory cells (especially eosinophils) and cellular elements
- airway hyper-responsiveness⁵
- intermittent airway narrowing (due to bronchoconstriction, congestion or oedema of bronchial mucosa or a combination of these) (see FIG. 73.1)

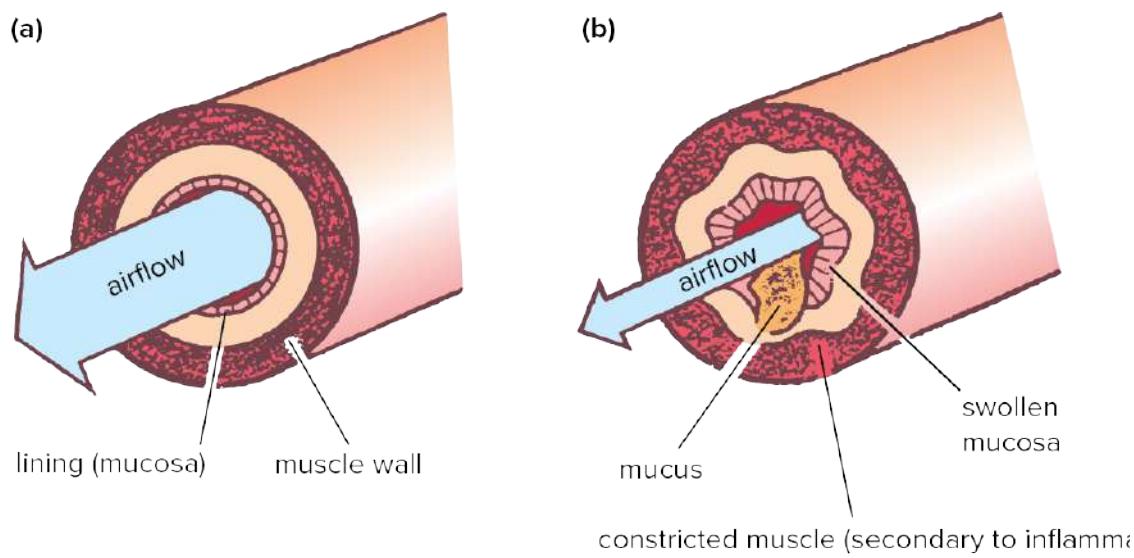


FIGURE 73.1 Airway changes in asthma: **(a)** normal airway, **(b)** airway in asthma

Causes of asthma

No single cause for asthma has been found, but a variety of factors may trigger an attack. These include specific factors such as viruses, allergens and non-specific factors such as temperature or weather changes and exercise. A checklist of *trigger factors* includes:

- A allergens—pollens, animal dander, dust mites, mould
- B bronchial infection
- C cold air, exercise
- D drugs—aspirin, NSAIDs, beta blockers
- E emotion, psychosocial problems—stress, laughter
- F food—sodium metabisulphite, seafood, nuts, monosodium glutamate
- G gastro-oesophageal reflux
- H hormones—pregnancy, menstruation
- I irritants—smoke, perfumes, smells
- J job—wood dust, flour dust, isocyanates, animals

Additional points

- Patients with asthma must never smoke.
- Atopic patients should avoid exposure to furred or feathered domestic animals if they have problems.
- About 90% of children with atopic symptoms and asthma demonstrate positive skin-prick responses to dust mite extract. Total eradication of house dust mite from the home is difficult.

Page 869

Clinical features

The classic symptoms are:

- wheezing
- coughing (chronic, esp. at night)
- tightness in the chest
- breathlessness

Asthma is likely if more than one of the above is present. Other supporting features:

- symptoms recurrent or seasonal

- worse at night or early morning
- history of allergies
- family history of asthma or allergies
- widespread audible wheeze on chest auscultation
- symptoms rapidly relieved by a short-acting beta agonist bronchodilator (SABA)

Note: Asthma should be suspected in children with recurrent nocturnal cough and in people with intermittent dyspnoea or chest tightness, especially after exercise.

Severe symptoms and signs are presented in the section on dangerous asthma later in this chapter.

Examination⁶

Physical signs may be present if the patient has symptoms at the time of examination.

The absence of physical signs does not exclude a diagnosis of asthma as the chest examination may be normal between attacks. During an attack, auscultation usually reveals diffuse, high-pitched wheezes throughout inspiration and most of expiration, which is usually prolonged. If wheeze is not present during normal tidal breathing it may become apparent during a forced expiration or after asking the child to exercise for 1–2 minutes. Wheeze does not necessarily indicate asthma.

Absence of wheeze in a breathless person is a serious sign.

Investigations

- Spirometry: a value of <75% for FEV₁/VC ratio indicates obstruction (a relatively accurate test and recommended for those who can perform it, i.e. most adults and children >6 years; see CHAPTER 38)
- Measurement of peak expiratory flow rate (PEFR) or spirometry before and after SABA: has a characteristic improvement >15% in FEV₁ and PEFR
- Bronchial provocation tests: airway reactivity is tested in a respiratory laboratory to inhaled histamine, methacholine or hypertonic saline (rarely required, but sometimes useful to confirm diagnosis)
- Fractional exhaled nitric oxide test
- An exercise challenge may also be helpful
- Allergy testing may be appropriate

- Chest X-ray: not routine but useful if complications suspected or symptoms not explained by asthma

Page 870

Significant historical advances in asthma management

1. The realisation that asthma is an inflammatory disease—the appropriate first- or second-line treatment in moderate to severe asthma is inhaled sodium cromoglycate (especially in children) or inhaled corticosteroids (ICS)
2. The regular use of spirometry
3. The use of spacers attached to inhalers/puffers
4. Improved and more efficient inhalers
5. Combined long-acting relievers and preventers including combinations of long-acting beta agonists (LABA) and ICS—the fixed-dose inhalers

Reasons for suboptimal asthma control are presented in TABLE 73.1 .

Table 73.1 Reasons for suboptimal asthma control^{2,4}

Poor compliance

Inefficient use of inhaler devices—poor technique

Procrastination in introducing optimal therapy

Failure to prescribe preventive medications, particularly inhaled corticosteroids, for chronic asthma

Using bronchodilators alone and repeating these drugs without proper evaluation

Reliance on inappropriate alternative therapies

Patient fears:

- concerns about corticosteroids
- overdosage
- developing tolerance
- embarrassment
- peer group condemnation

Doctor's reluctance to:

- use corticosteroids

- recommend obtaining a mini peak flow meter
- recommend obtaining a spacer

Medical factors:

- obesity
 - rhinosinusitis
 - GORD
 - sleep apnoea
 - vocal cord dysfunction
 - smoking/COPD
-

Measurement of peak expiratory flow rate

Patients with moderate to severe chronic asthma require regular measurement of PEFR, which is more useful than subjective symptoms in assessing asthma control. This allows the establishment of a baseline of the ‘best effort’, monitors changes and allows the assessment of asthma severity and response to treatment.

Spirometry including FEV₁ is the gold standard (see [CHAPTER 38](#)). Peak flow meters are not a substitute for spirometry, as there is considerable variation between users and instruments. However, PEFR has a place in helping patients self-manage their asthma by comparing the current result with their best peak flow.

Spacers⁷

Large volume spacers

Metered dose inhalers (MDIs) are convenient, but usually deliver less drug to the lungs than when a spacer is fitted onto the mouthpiece of the inhaler. One puff at a time is put in the spacer. The patient breathes in from its mouthpiece, taking 1–2 very deep breaths, or 4–6 normal breaths (especially in children). Spacers are particularly useful for adults having trouble with the MDI and for younger children (but older than 3 years). Spacers are very efficient, overcome poor technique and cause less irritation of the mouth and throat (see [FIG. 73.2](#)).

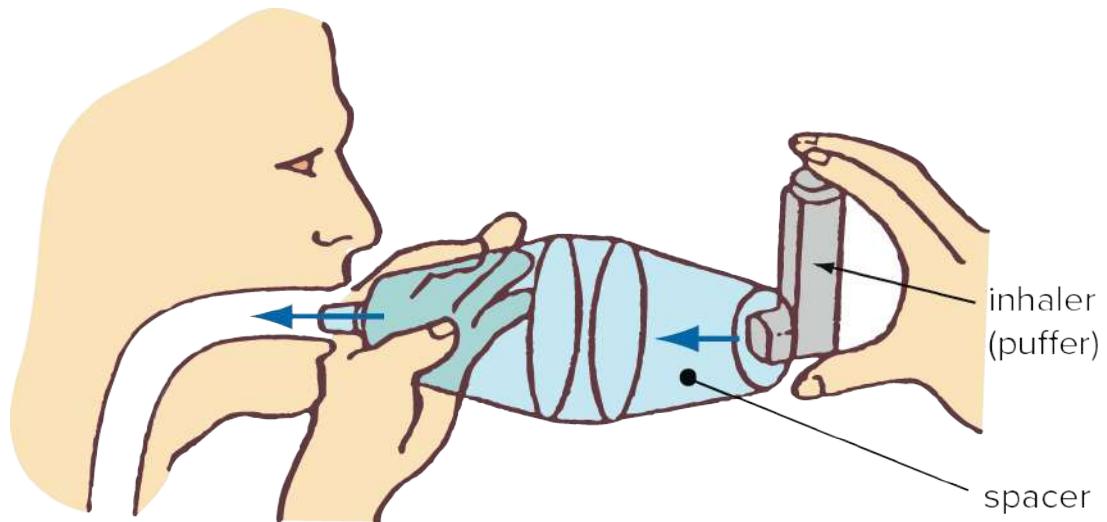


FIGURE 73.2 Using a spacer device. Rules: children—single puff, then 4–5 breaths; adults—single puff, 1–4 breaths.

They allow increased airway deposition of inhalant and less oropharyngeal deposition.

Note: It is recommended to dip plastic spacers into water with ordinary household detergent and dry in sunlight (no rinsing, no wiping) every 10 days or at least monthly.

Small volume spacers

Children under 5–6 years and/or 20 kg can use an MDI and a small volume valved spacer (AeroChamber, Breath-A-Tech) with a face mask.

Management principles

Page 871

Starting treatment¹

- Confirm the diagnosis.
- Assess recent asthma control and risk factors (see TABLE 73.2).

Table 73.2 Asthma severity classification for an untreated newly diagnosed adult with asthma ⁸
--

Lung function	Estimated starting daily dose range of
---------------	--

Severity/grade	Status before treatment	FEV ₁ or PEFR (% predicted)	Recommended β ₂ -agonist	ICS required to achieve good control
Intermittent	Episodic Symptoms < weekly Night symptoms <2 per month Mild occasional symptoms with exercise	≥80%	SABA prn	Regular ICS not required Add preventer if ≥3 uses of SABA/week
Mild persistent	Symptoms > weekly, not every day Night symptoms >2 per month Symptoms regularly with exercise	≥80%	SABA prn	<250 mcg beclomethasone <400 mcg budesonide <250 mcg fluticasone <160 mcg ciclesonide Increase dose if >2 SABA 2–3 times daily
Moderate persistent	Symptoms every day Night symptoms > weekly Several known triggers apart from exercise	60–80%	LABA + SABA prn	250–400 mcg beclomethasone 400–800 mcg budesonide 250–500 mcg fluticasone 160–320 mcg ciclesonide
Severe persistent	Symptoms every day Wakes frequently at night with cough/wheeze Chest tightness on	<60%	LABA + SABA prn	>400 mcg beclomethasone >800 mcg budesonide >500 mcg fluticasone >320 mcg ciclesonide

waking
Limitation of
physical
activity



- If moderately severe inflammatory airways disease is not treated (with inhaled corticosteroids) there is the risk of fixed irreversible airways obstruction from submucosal fibrosis.
- Choose initial treatment appropriate to the above.
- Avoid the use of LABA on its own (i.e. without ICS), as it is associated with an increased risk of asthma death.
- Document evidence in notes.
- Identify management goals in collaboration.
- Provide clear written patient information.
- Educate and review regularly.

Goals of management:

- absent or minimal daytime symptoms and no nocturnal symptoms; restore normal airway function (>80% of predicted)
- maintain best possible lung function at all times—keep asthma under control
- reduce morbidity
- control asthma with the use of regular anti-inflammatory medication and relieving doses of β_2 -agonist when necessary

Long-term goals:

- achieve use of the least drugs, least doses and least side effects
- reduce risk of fatal attacks
- reduce risk of developing irreversible abnormal lung function

Page 872

Good asthma control

- Minimal symptoms day and night

- No nocturnal waking due to asthma
- No limitation of normal activity or exercise
- Minimal need for reliever medication
- No exacerbations
- Normal or near-normal lung function (FEV_1 and/or $\text{PEFR} >80\%$ of predicted or best)
- No side effects of medication

Questions to assess asthma control¹

Ask about:

- limitation of daily activities
- shortness of breath
- sleep disturbance
- use of reliever medication
- perceived level of asthma control

Pharmacological agents to treat asthma

Simple classification

- Reliever = bronchodilator
- Preventer = anti-inflammatory
- Symptom controller = long-acting β_2 -agonist (LABA)

It is useful to teach patients the concept of the ‘preventer’ and the ‘reliever’ for their asthma treatment. The pharmacological treatment of asthma is summarised in TABLE 73.3 .

Generic types	Examples	Nebulising solution	Oral	Vehicle of administration		
				Aerosol (metered dose inhalation)	Dry powder (inhalation)	Injection
Salbutamol	Ventolin	✓	✓	✓	✓	✓
Salmeterol	Serevent			✓	✓	
Terbutaline	Bricanyl	✓	✓		✓	✓
Eformoterol	Foradile, Oxis				✓	
Indacaterol	Onbrez					
Adrenaline						✓
Ipratropium bromide	Atrovent	✓		✓		
Theophylline	Nuelin			✓		
Aminophylline						✓
Sodium cromoglycate	Intal	✓		✓	✓	
Nedocromil sodium	Tilade			✓		
Beclomethasone	QVAR			✓	✓	
Budesonide	Pulmicort	✓		✓	✓	
Ciclesonide	Alvesco			✓		
Fluticasone	Flixotide	✓		✓	✓	
preventer (ICS)—fixed combination MDI tide)				✓	✓	
form)				✓		
nbicort)				✓	✓	
lloptia)					✓	
				✓		

'Preventer' drugs: anti-inflammatory agents⁸

These medications are directed towards the underlying abnormalities—bronchial hyper-reactivity and associated airway inflammation. Treatment with a 'preventer' is recommended if asthma episodes are >3/week or those who use SABA >3 times a week.

Corticosteroids

Inhaled (ICS)

Types:

- beclomethasone
- budesonide
- ciclesonide (single daily dose)
- fluticasone

Dose range:

- microgram ranges differ according to drug; aim for lowest effective dose

Availability:

- MDI
- Turbuhaler
- Autohaler
- Accuhaler

Frequency:

- once or twice daily (adherence may be greater with once daily)

Side effects:

- oropharyngeal candidiasis, dysphonia (hoarse voice)—less risk with once daily ciclesonide
- bronchial irritation: cough
- adrenal suppression (doses of 2000 mcg/daily; sometimes as low as 800 mcg)

Note: Rinse mouth with water and spit out after using inhaled steroids.

ICSS have a flat dose-response curve so there are diminishing returns in prescribing above beclomethasone or budesonide 1000 mcg/day or fluticasone 500 mcg/day. For newly diagnosed patients with mild-to-moderate asthma ‘start low and step up prn’ (e.g. 250–400 mcg/day).⁶ Step down the dose when safe to do so.

Note: Most adults and older adolescents with asthma should be on long-term inhaled corticosteroid therapy.¹

Oral

Prednisolone is used mainly for exacerbations. It is given with the usual inhaled corticosteroids and bronchodilators.

Dose:

- up to 1 mg/kg/day (usual max. 50 mg) for 3 days to 2 weeks

Side effects:

- these are minimal if drug is used for short periods
- long-term use has significant side effects: osteoporosis, glucose intolerance, adrenal suppression, thinning of skin and easy bruising

Note: Short-term oral corticosteroids can be ceased abruptly without tapering. Clinical trials are tending to favour shorter courses.

Cromones

These are sodium cromoglycate (SCG) and nedocromil sodium. SCG is available as dry capsules for inhalation, metered dose aerosols and a nebuliser solution. They are often inhaled via a spacer in children. Adverse effects are uncommon; local irritation may be caused by the dry powder. Systemic effects do not occur.

Nedocromil is used for frequent episodic asthma in children over 2 years of age for the prevention of exercise-induced asthma and the treatment of mild-to-moderate asthma in some adults. The initial dose is 2 inhalations qid. Adverse effects are uncommon.

Leukotriene antagonists

These drugs (in Australia, primarily montelukast) are very useful for seasonal asthma and aspirin-sensitive asthma and reduce the need for inhaled steroids or offer an alternative for those who cannot tolerate ICSS or have trouble using an inhaler. Favourable evidence is based on a small number of trials only, mostly in children but some adults benefit.⁹ Montelukast is taken as a 5 or 10 mg chewable tablet once daily.

Indications for preventive therapy¹⁰

Guidelines for introducing preventive asthma therapy in adults and children include any of the following:

- requirement of β_2 -agonist >2 days per week or >1 canister every 3 months (excluding pre-exercise)
- symptoms (non-exercise) >2 times per week between attacks
- any symptoms during the night or on waking
- spirometry showing reversible airflow obstruction during asymptomatic phases
- asthma significantly interfering with physical activity despite appropriate pre-treatment
- asthma attacks \geq twice per month
- infrequent asthma attacks but severe or life-threatening

'Reliever' drugs or bronchodilators

The three groups of bronchodilators are:

- the β_2 -adrenoceptor agonists (β_2 -agonists)—short acting (SABA) and long acting (LABA)
- methylxanthines—theophylline derivatives
- anticholinergics

β_2 -agonists

These drugs stimulate the β_2 adrenoreceptors and thus relax bronchial smooth muscle. Inhalation is the preferred route of delivery; the vehicles of administration include metered dose inhalation, a dry powder, and nebulisation where the solution is converted to a mist of small droplets by a flow of oxygen or air through the solution.

Oral administration of β_2 -agonists is not recommended. The inhaled drugs produce measurable bronchodilation in 1–2 minutes and peak effects by 10–20 minutes. The traditional agents such as salbutamol and terbutaline are short-acting preparations. The new longer-acting agents (LABA) include salmeterol, eformoterol and vilanterol.

LABAs should always be used in combination with an ICS, not as monotherapy.¹¹

Theophylline derivatives

These oral drugs may have complementary value to the inhaled agents but tend to be limited by side effects and efficacy.

Anti-IgE monoclonal antibodies

These newer agents (e.g. omalizumab) bind IgE without activating mast cells. They are directed for use in patients >12 years of age with moderate to severe allergic eosinophilic asthma who have been treated by ICS and who have raised serum IgE levels. They are given by SC injection and the PBS stipulates specialist initiation.

Antibiotic use for chronic asthma¹²

Antibiotics are not recommended apart from clinical evidence of super-respiratory infection. Trials of daily oral azithromycin have provided weak evidence of possible benefit in reducing exacerbations of asthma and COPD, but this has not been recommended in clinical practice.¹³

Starting treatment

Current treatment supports the initial treatment (summarised in TABLE 73.2) of a SABA with low to moderate doses of ICS with estimated equivalent doses shown in the table.

Initiate therapy sufficient to achieve best lung function promptly.

Wean inhaled corticosteroids to the minimum dose needed to maintain adequate asthma control.

Prophylactic agents

This term is reserved for those medications that are taken prior to known trigger factors, particularly for exercise-induced asthma.

Exercise-induced asthma (options)

- β_2 -agonist inhaler (puffer): two puffs 5 minutes immediately before exercise last 1–2 hours. LABA such as salmeterol and eformoterol are more effective if used with ICS.
- SCG or nedocromil, two puffs
- Combination β_2 -agonist + SCG (5–10 minutes beforehand)
- Montelukast 10 mg (less in children ≥ 2 years) oral daily or 1–2 hours beforehand
- Paediatricians often recommend a non-drug warm-up program as an alternative to medication.

Ongoing management

The three-step asthma control plan^{14,15}

The National Asthma Council of Australia has developed the following follow-up plan, summarised in [FIGURE 73.3](#).

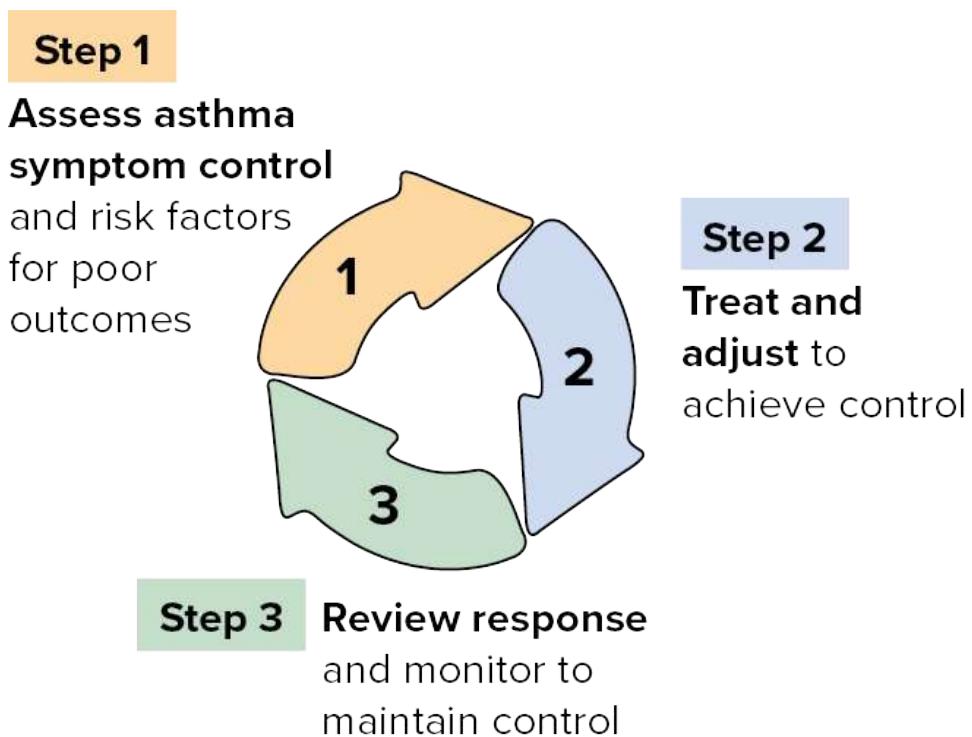


FIGURE 73.3 Asthma: ongoing management steps

Source: Reproduced with permission from NPS Medicine Wise

Step 1: Assess asthma symptom control and identify the patient's risk factors.

- Assess asthma symptom control over the previous 4 weeks.
- Assess the patient's risk factors.
- Exclude factors contributing to poor control before intensifying preventer treatment:
 - check adherence
 - check inhaler technique
 - check inhaler device is appropriate
 - consider that symptoms may be due to alternative or comorbid diagnoses

Page 875

Step 2: Treat and adjust to achieve good control.

- All patients should have a reliever inhaler for as-needed use.

- Most can achieve well-controlled asthma with low-dose ICS.
- Trial low-dose ICS before ICS/LABA fixed combination therapy.¹⁶
- Reserve ICS/LABA as a later option. This combination is too readily used in Australia.
- Where appropriate, step down treatment.
- Schedule follow-up visit.

See [TABLE 73.3](#) .

Step 3: Review response and monitor to maintain control.

- Review diagnosis and treatment regularly.
- Monitor to maintain control.

A general management plan for chronic asthma is summarised in [FIGURE 73.4](#) .

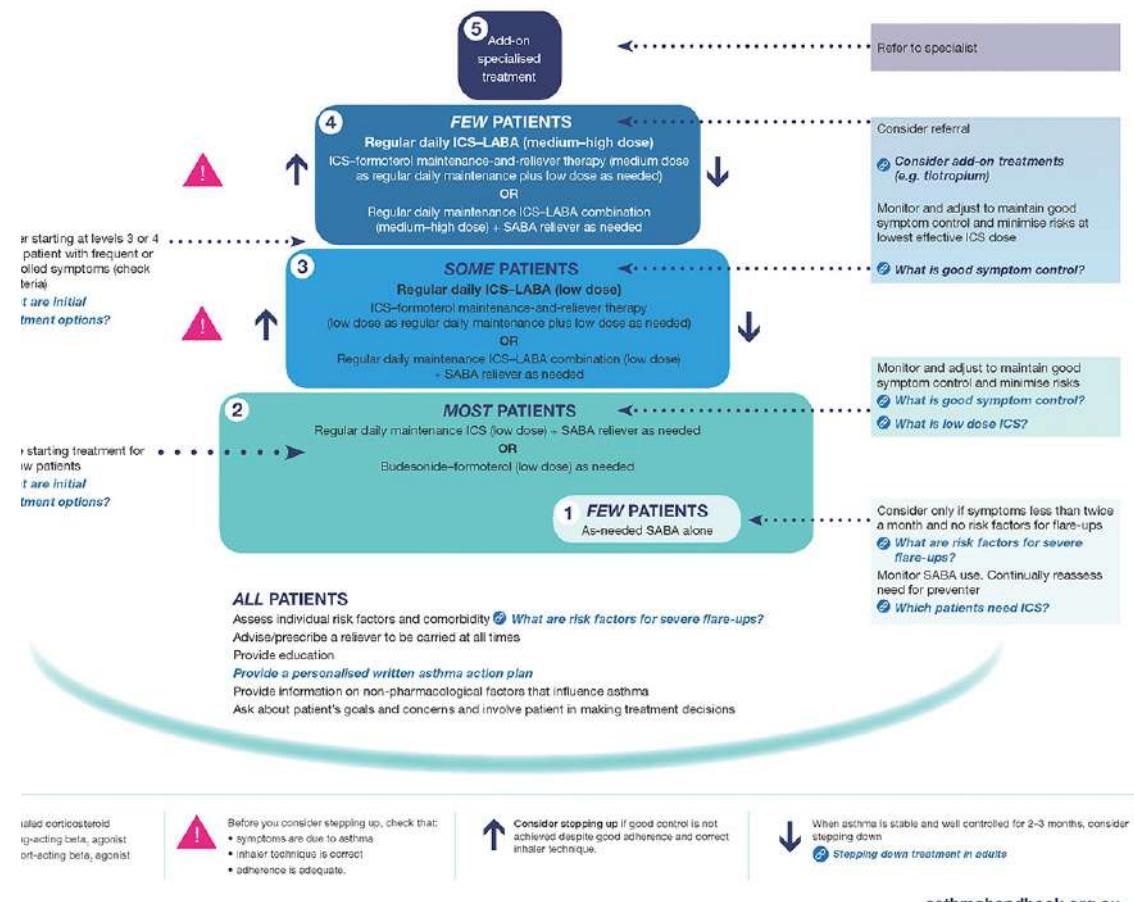


FIGURE 73.4 Stepped approach to adjusting asthma medication in adults

This figure comes from the National Asthma Foundation Australia and is based on Australian Treatment Standards and medications available in Australia and not designed or intended for international use.

Practice tips

- For breakthrough asthma or persistent poorly controlled asthma with poor compliance switch to combined medication (e.g. Seretide MDI Accuhaler or Symbicort).

Correct use of the asthma MDI (puffer)

Did you know that:

- faulty inhaler technique occurs in at least one-third of users?
- in faulty technique, up to 90% of the medication sticks to the mouth and does not reach the lungs?
- it is the inhalation effort—not the pressure from the aerosol—that gets the medication to the lungs?
- it is important to instruct patients properly and check their technique regularly?

The two main techniques

The open-mouth technique and the closed-mouth technique are the main methods, and both are effective but the closed-mouth technique is preferred. Both techniques are suitable for most adults. Most children from the age of 7 can learn to use puffers quite well.

The closed-mouth technique

See [FIGURE 73.5](#).



FIGURE 73.5 Using the metered dose inhaler: the closed-mouth technique

Instructions for patients:

1. Remove the cap. Shake the puffer vigorously for 1–2 seconds. Hold it upright (canister on

top).

2. Place the mouthpiece between your teeth (do not bite it) and close your lips around it.
3. Breathe out slowly and gently to a comfortable level.
4. Tilt your head back slightly with your chin up.
5. Just as you then start to breathe in (slowly) through your mouth, press the puffer firmly, once. Breathe in as far as you can over 3–5 seconds. (Do not breathe in through your nose.)
6. Remove the puffer from your mouth and hold your breath for about 10 seconds; then breathe out gently.
7. Breathe normally and then repeat the inhalation if you need to.

Page 876

Extra points

- The usual dose of standard MDI is one or two puffs (adult) every 3–4 hours for an attack (four puffs in children).
- If you do not get adequate relief from your normal dose, contact your doctor.
- It is quite safe to increase the dose, such as to 4–6 puffs.
- If you are using your inhaler very often, it usually means your other asthma medication is not being used properly.

Autohaler

The Autohaler is a breath-activated MDI which can improve lung deposition in patients with poor inhaler technique.

Turbuhaler

The Turbuhaler is a dry powder delivery system that is widely used as an alternative to the MDI. It is a breath-activated device.

Other dry powder devices are the Accuhaler and Diskhaler.

Spacers versus nebulisers

Both MDIs via a spacer and dry powder inhalers are at least as effective as a nebuliser for treating acute exacerbations in both adults and children.¹⁷ They are considerably cheaper and more readily available than an electrically powered device.

Summary of devices

- Breath-activated MDIs: Autohaler
- Breath-activated dry powder inhalers: Accuhaler, Aerolizer, Diskhaler, Rotahaler, Spinhaler, Turbuhaler
- Large volume spacer: Nebuhaler, Volumatic
- Small volume spacer: Aerochamber, Breath-A-Tech

Dangerous asthma

Failure to recognise the development of a severe attack has cost the lives of many asthmatics. A severe attack can start suddenly (even in mild asthmatics) and catch people by surprise.

High-risk patients

People who have experienced one or more of the following are more likely to have severe attacks:

- previous severe asthma attack
- previous hospital admission, especially admission to intensive care
- hospital attendance in the past 12 months
- long-term oral steroid treatment
- carelessness with taking medication
- night-time attacks, especially with severe chest tightness
- recent emotional problems
- frequent SABA use

Early warning signs of a severe asthma attack:

- symptoms persisting or getting worse despite adequate medication
- increased coughing and chest tightness
- poor response to two inhalations
- benefit from inhalations not lasting 2 hours

- increasing medication requirements
- sleep being disturbed by coughing, wheezing or breathlessness
- chest tightness on waking in the morning
- low PEFR readings

Thunderstorm asthma¹⁸

A likely mechanism is that this occurs when the high winds preceding a storm whip up massive quantities of rye grass pollens which absorb moisture and rupture, releasing allergens. People most vulnerable to an asthma attack are those with a history of allergy and poorly controlled asthma in particular. When an attack is imminent, at-risk people should stay inside with windows and doors closed, take preventer medication, follow action plans and ensure reliever drugs are readily available. Treatment is with a salbutamol spray followed by corticosteroids.

Page 877

Seasonal allergic rhinitis patients not on constant intranasal corticosteroids (INCS) should start taking these before the pollen season and continue until pollen levels abate. For those with asthma include a combination of an INCS and intranasal antihistamine if symptoms are severe or not controlled by INCS alone.¹

Dangerous signs

- Marked breathlessness, especially at rest
- Sleep being greatly disturbed by asthma
- Asthma getting worse quickly rather than slowly, despite medication
- Feeling frightened
- Difficulty in speaking; unable to say more than a few words
- Pulsus paradoxus
- Exhaustion and sleep deprivation
- Drowsiness or confusion
- Chest becoming ‘silent’ with a quiet wheeze, yet breathing still laboured
- Cyanosis
- Chest retraction
- Respiratory rate greater than 25 (adults) or 50 (children)

- Pulse rate >120 beats/min
- Peak flow <100 L/min or <40% predicted FEV₁
- Oximetry on presentation (SaO₂) <90%

Asthma action plans

Examples of action plans for patients are presented below. Having a written asthma action plan as part of self-management reduces asthma-related mortality and morbidity.¹⁹

Action plan

If you are distressed with severe asthma:

- call an ambulance and say ‘severe asthma attack’ (best option)

or

- call your doctor

or

- if you are having trouble finding medical help, get someone to drive you to the nearest hospital

Follow the ‘4 × 4 × 4’ plan with your reliever medication, but keep using it continuously if you are distressed.

Keep an asthma action plan on a card for easy reference. Remember to have extra prednisolone and salbutamol in a household where you may be staying.

Asthma first-aid action plan

Name _____

Contacts:

Dr _____ Tel _____

Ambulance tel (000)

1. Sit upright and stay calm.
2. Take 4 separate puffs of a reliever puffer (one puff at a time) via a spacer device. Just use the puffer on its own if you don’t have a spacer. Take 4 breaths from the

spacer after each puff

3. Wait 4 minutes. If there is no improvement, take another 4 puffs. (The $4 \times 4 \times 4$ rule)
4. If little or no improvement CALL AN AMBULANCE IMMEDIATELY (dial 000) and state that you are having an asthma attack. Keep taking 4 puffs every 4 minutes until the ambulance arrives.

See your doctor immediately after a serious asthma attack.

The acute severe asthma attack

Summary (adult dosage):^{6,8}

- continuous nebulised salbutamol (or terbutaline) with oxygen flow rate 6–8 L/min if nebuliser available (or 12 puffs of β_2 -agonist inhaler, with spacer, using one loading puff at a time followed by 4–5 normal tidal breaths)

Ipratropium bromide may be mixed with β_2 -agonist for concurrent nebulisation.

- parenteral β_2 -agonist (e.g. salbutamol 500 mcg IM, SC)
- corticosteroids, e.g. prednisolone 50 mg (o) statim then daily until resolved

or

- hydrocortisone 250 mg IV or IM 6 hourly
- oxygen 8 L/min by face mask to maintain $\text{SpO}_2 > 92\text{--}95\%$ or at least 95% in children
- monitor PEFR

Further deterioration:

- magnesium sulphate 25–100 mg/kg (max. 2 gm) IV over 20 min
- adrenaline 0.5 mg 1:1000 SC, IM or 1:10 000 IV

Guidelines for spacer use in severe asthma⁸

- Frequency—every 20 minutes (first hour)

- One puff actuation at a time
- 4–5 normal breaths each time
- 25 kg or <6 years:
 - 6 puffs—salbutamol
 - 2 puffs—ipratropium
- 25–35 kg:
 - 8 puffs salbutamol
 - 3 puffs ipratropium
- >35 kg:
 - 12 puffs—salbutamol
 - 4 puffs—ipratropium
- For moderate asthma use salbutamol only

The management of severe asthma is presented in [CHAPTERS 89](#) and [120](#).

Asthma in children

The prevalence of asthma is increasing in childhood and the management (especially in infants) is always a concern for the family doctor. The aim of treatment is to enable children to enjoy a normal life, comparable with that of non-asthmatic children, with the least amount of medication and at minimal risk of adverse events. Maintenance should be determined by symptom control and lung function, especially using clinical criteria since PEFR is unreliable. A diagnosis of asthma should not be made if cough is the only or predominant symptom and no signs of airflow limitation.

Key checkpoints

- Seek specialist advice for children under 6 months of age.
- Bronchodilators, inhaled or oral, are ineffective under 12 months.
- The delivery method is a problem in children and [TABLE 73.4](#) gives an indication of what systems can be used at various levels.

Table 73.4 Delivery systems for asthma in children

Vehicle of administration	Age in years			
	Under 2	2–4	5–7	8 and over
MDI (puffer) alone			*	✓
MDI + small volume spacer** + face mask	✓	✓		
MDI + large volume spacer**		✓	✓	✓
Nebuliser/air compressor/face mask	✓	✓	✓	✓
Dry powder inhalers (e.g. Turbuhaler, Rotahaler)			*	✓
Breath-activated device			*	✓

*Possible in some individual children

**Small volume spacer—2 tidal breaths; large volume—3

- In the very young (e.g. 1–2 years old), a spacer with a face mask such as Aerochamber or Breath-A-Tech can deliver the aerosol medication.
- The PEFR should be measured in all asthmatic children older than 6 years. Children under 6 years generally cannot cope with the meters and those with mild asthma don't usually need PEFR measurement.
- The Turbuhaler is usually not practical under 7–8 years.

Prophylaxis in children

The non-steroidal medications, montelukast (oral) and SCG and/or nedocromil sodium by inhalation, are the prophylactic drugs of choice in childhood chronic asthma of mild-to-moderate severity.

If there is no clinical response to these agents in 4 weeks, consider use of inhaled corticosteroids, but the risks versus benefits must always be considered. Any dose equal to or greater than 400 mcg in children can have side effects, including growth suppression and adrenal suppression. Aim for a maintenance of 100–400 mcg, which keeps the child symptom-free. Once this stage is reached, consider stopping treatment or changing to the non-steroidal options.

Leukotriene antagonists taken orally for children aged 6 years and above is another option.

Delivery systems for children are presented in TABLE 73.4. Guidelines for the management of asthma in children are summarised in TABLE 73.5 .

Table 73.5 Stepwise interval management plan for children^{8,17}

Grade of asthma	Therapeutic agents
Mild —infrequent episodic:	SABA prn
<ul style="list-style-type: none"> • attacks not severe • >6–8 weeks apart 	
Moderate —frequent episodic:	SABA prn <i>and</i> (trial of) <ul style="list-style-type: none"> • montelukast especially 2–5 yo: 4 mg (o) nocte 6–14 yo: 5 mg (o) nocte <i>or</i> <ul style="list-style-type: none"> • cromolyn stepped-up regular preventer • ICS—minimum effective dose, e.g. beclomethasone 100–200 mcg/day budesonide 200–400 mcg/day
Severe —persistent asthma:	Referral SABA prn <i>and</i> <ul style="list-style-type: none"> • ICS (as above) • consider combination LABA + ICS (>6 yo) Add: <ul style="list-style-type: none"> • ipratropium bromide (nebuliser) • oral prednisolone (when required)

When to refer

- If you are doubtful about the diagnosis
- For advice on management when asthmatic control has failed or is difficult to achieve

Practice tips

- Reassure the patient that 6–10 inhaled doses of a β_2 -agonist is safe and appropriate for a severe attack of asthma.
- It is important to achieve a balance between undertreatment and overtreatment.
- Beware of patients, especially children, manipulating their peak flow meter results.
- Get patients to rinse out their mouth with water and spit it out after inhaling corticosteroids.
- Patients who are sensitive to aspirin/salicylates need to be reminded that salicylates are present in common cold cure preparations and agents such as Alka-Seltzer.²
- Aspirin-sensitive asthma usually manifests late in life with associated rhinitis. It cross-sensitises with NSAIDs.
- Possible side effects of inhaled drugs can be reduced by always using a spacer with the inhaler, using the medication qid rather than bd, rinsing the mouth, gargling and spitting out after use, and using corticosteroid-sparing medications.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Asthma
- Asthma: correct use of your aerosol inhaler
- Asthma: dangerous asthma

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74 Chronic obstructive pulmonary disease

Tobacco drieth the brain, dimmeth the sight, vitiateth the smell, hurteth the stomach, destroyeth the concoction, disturbeth the humors and spirits, corrupteth the breath, induceth a trembling of the limbs, exsiccateth the windpipe, lungs and liver, annoyeth the milt, scorcheth the heart, and causeth the blood to be adusted.

TOBIAS VENNER (1577–1660), *VIA RECTA AD VITAM LONGAM*

Chronic obstructive pulmonary disease (COPD) is described by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as a:

common, preventable and treatable disease characterised by non-fully reversible persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. It is basically a structural disease. Exacerbations and comorbidities contribute to the overall severity in individual patients.¹

COPD typically affects middle-aged and older people with the usual age of onset in the fifth and sixth decades. It is the fourth leading cause of death and the third leading burden of disease in Australia, affecting 12.4% of Australians between 45 and 70 years. Early diagnosis and treatment is important to outcome and the provisional diagnosis should be based on the clinical features of breathlessness and cough in a smoker or ex-smoker.

Cigarette smoking is undoubtedly the major cause of both chronic bronchitis and emphysema, although only 10–15% of smokers develop the diseases.² Chronic bronchitis is defined as >3 episodes per year for 2 years while emphysema is destruction of alveoli. The clinical differences between asthma and bronchitis are summarised in [CHAPTER 38](#). Patients experiencing overlap of asthma and COPD should be identified and treated differently to patients with either condition alone.

Lifelong passive smoking exposure increases COPD risk by 2.2 to 4.0 times.

[FIGURE 74.1](#) illustrates the influence of smoking on lung function.

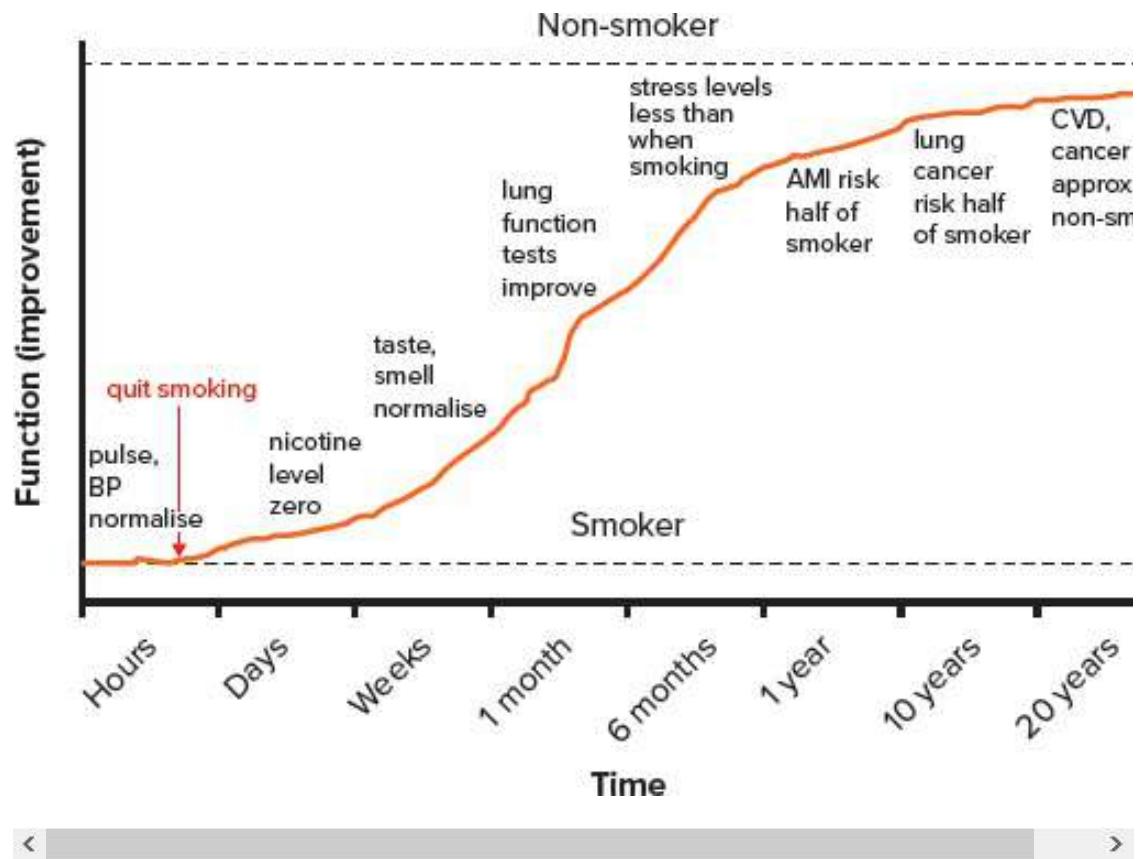


FIGURE 74.1 Clinical trajectory of a person who quits smoking

Factors in causation

- Cigarette smoking (usually 20/day for 20 years or more)⁴
- Natural fuel—wood, twigs, crop residue
- Air pollution (outdoor and indoor)
- Airway infection/chronic bronchitis
- Occupation: related to cadmium, silica, dusts
- Familial factors: genetic predisposition
- Alpha₁-antitrypsin deficiency (emphysema)
- Bronchial hyper-responsiveness

Diagnosis and management of COPD

The COPDX Plan guidelines⁵ developed by the Australian Lung Foundation and the Thoracic Society of Australia and New Zealand provide an appropriate framework for diagnosis and management. The key recommendations are: Confirm diagnosis, Optimise function, Prevent deterioration, Develop a self-management plan and manage exacerbations.

Page 882

C—Confirm diagnosis and assess severity

Symptoms

- Breathlessness
 - Cough
 - Sputum production
- }
- Chest tightness
 - Wheezing
 - Airway irritability
- Fatigue
- Anorexia
- Weight loss
- }
- main symptoms
- with advanced disease

Consider the diagnosis of COPD in all smokers and ex-smokers older than 35 years. The diagnosis of COPD rests on the demonstration of airflow obstruction.

The sensitivity of the physical examination for detecting mild to moderate COPD is poor.

Signs

The signs vary according to the nature of the disease and the presence of infection. Signs may be completely absent in the early stages of COPD: chronic bronchitis may present only with wheezing, while dyspnoea is a feature of associated airflow obstruction.

Signs may include:

- tachypnoea
- reduced chest expansion
- hyperinflated lungs
- hyper-resonant percussion
- diminished breath sounds ± wheeze
- ‘pink puffer’—always breathless
- ‘blue bloater’—oedematous and central cyanosis
- signs of respiratory failure
- signs of cor pulmonale

The diagnosis is usually clinical with a history of increasing dyspnoea and sputum production in a lifetime smoker with no (or minimal) features of asthma. It is imprudent to make a diagnosis of chronic bronchitis and emphysema in the absence of cigarette smoking unless there is a family history suggestive of alpha₁-antitrypsin deficiency.⁶

At diagnosis, up to 50% of lung function may have been lost.

Investigations

Pulmonary function tests

Spirometry remains the gold standard for diagnosing, assessing and monitoring COPD. Post-bronchodilator forced expiratory ratio (FER) <0.7 is required for diagnosis.

Definition

COPD Post-bronchodilator FEV₁/FVC of <0.70 (<70%) and FEV₁ <80% predicted.

The Australian stages of severity based on FEV₁% predicted are mild (60–80%), moderate (40–50%) and severe (<40%), while the GOLD¹ staging is 1. mild ($\geq 80\%$), 2. moderate (50–80%), 3. severe (30–50%), 4. very severe (<30%).

Chest X-ray

This can be normal (even with advanced disease) but characteristic changes occur late in disease. May exclude lung cancer >1 cm.

Blood gases

- May be normal
- $\text{PaCO}_2 \uparrow$; $\text{PaO}_2 \downarrow$ (advanced disease)

Gas transfer factor

- A reduced diffusing capacity of the lungs for carbon monoxide (DLCO) is a feature of COPD. DLCO is not reduced in asthma.

ECG

- This may show evidence of cor pulmonale

Sputum culture

- If resistant organism suspected

FBE

- To identify anaemia and polycythaemia
- Haemoglobin and PCV may be raised in COPD

O—Optimise function

The principal goals of therapy are to stop smoking, to optimise function through relief of symptoms with medication and pulmonary rehabilitation, and to prevent or treat aggravating factors and complications.

TABLE 74.1 is a useful consultation checklist mnemonic.⁷

Table 74.1 SMOKEs, a consultation checklist for chronic obstructive pulmonary disease⁷

S = Smoking cessation

M = Medication—inhaled bronchodilator, vaccines (influenza, pneumococcus), corticosteroids (if indicated)

O = Oxygen—is it needed?

K = ‘K’omorbidity—cardiac dysfunction, sleep apnoea, osteoporosis, depression, asthma, GORD

E = Exercise and rehabilitation

S = Surgery—bullectomy, lung volume reduction surgery, single-lung transplantation

Long-term treatment

Advice to patient

- If you smoke, you must stop (persuading the patient to stop smoking is the key to management). The only treatment proven to slow the progression of COPD is smoking cessation.³ Consider smoking cessation medications.
- Avoid places with polluted air and other irritants, such as smoke, paint fumes and fine dust.
- Go for walks in clean, fresh air.
- A warm, dry climate is preferable to a cold, damp place (if prone to infections).
- Get adequate rest.
- Avoid contact with people who have colds or flu.
- Optimal diet—reduce weight if necessary.

Physiotherapy

Refer to a physiotherapist for chest physiotherapy, breathing exercises and an aerobic physical exercise program.

Drug therapy⁶

In the long-term treatment of COPD, bronchodilators are recommended for the relief of wheezing and shortness of breath. Bronchodilation is important to allow ‘lung emptying’ and reduce gas trapping. These include: short-acting β_2 -agonists (SABAs, e.g. salbutamol, terbutaline) and short-acting anticholinergic drugs (ipratropium bromide); long-acting β_2 -agonists (LABAs, e.g. eformoterol, salmeterol, indacaterol, vilanterol); long-acting anticholinergic drugs with muscarinic antagonist action (LAMAs, e.g. tiotropium, glycopyrronium, umeclidinium) which block parasympathetic bronchial constriction; and corticosteroids.

The preferred route of administration of bronchodilator is by inhalation, which requires correct device technique.

Inhaled drugs can be administered by MDIs, dry powder devices or nebulisers. The evidence suggests that an MDI and spacer are as effective as a nebuliser—they are also simpler and

cheaper—but the appropriate method depends on patient needs and preference.

The usefulness of a bronchodilator for an individual can only be assessed by a therapeutic trial, accepting either objective improvement in lung function or improvement in symptom control as endpoints. Individual adherence and preference play big roles in treatment decisions over time.

Short-acting bronchodilator therapy

Most studies suggest that short-acting β_2 -agonists and ipratropium bromide are equally efficacious in patients with COPD. If patients do not respond adequately to one of these bronchodilators then it is appropriate to consider a trial of a combination of the two classes of bronchodilator with objective monitoring of response.

Use the following by inhalation:⁶

salbutamol 100–200 mcg, up to 4 times daily

or

terbutaline 500 mcg, up to 4 times daily

or (with and without)

ipratropium bromide 40–80 mcg, up to 4 times daily

For patients unable to use an MDI with a spacer or any other handheld device inhalation, a nebuliser should be used, with the following doses:

salbutamol or terbutaline 2.5 to 5 mg

and/or

ipratropium bromide 250 to 500 mcg by nebuliser, up to 4 times a day

Long-acting bronchodilator therapy

Long-acting β_2 -agonists can be used in patients who remain symptomatic despite treatment with combinations of short-acting bronchodilators and those with frequent exacerbations. Used regularly, they can be effective and may be more convenient than using short-acting bronchodilators.⁸ Long-acting anticholinergic therapy² with tiotropium bromide (taken by inhalation) has been proven to reduce the frequency of exacerbations with COPD compared with short-acting anticholinergic drugs. The choice of drug can be determined by the patient's response to a trial of the drug, the drug's adverse effects and cost including PBS listing.

For treatment with long-acting bronchodilator by inhalation, see TABLE 74.2 .

Page 884

Table 74.2 Long-acting bronchodilators⁶

LABAs*

eformoterol	12 mcg twice daily
salmeterol	50 mcg twice daily
indacaterol	150–300 mcg daily
olodaterol	5 mcg daily

LAMAs**

aclidinium	322 mcg twice daily
tiotropium	18 mcg daily
glycopyrronium	50 mcg daily
umeclidinium	62.5 mcg daily

*LABA = long-acting β_2 -agonist

**LAMA = long-acting muscarinic antagonist

Corticosteroids⁶

Only 10% of patients with stable COPD benefit in the short term from inhaled corticosteroids (ICS). There are no distinguishing clinical features to predict in advance which patients may respond. The aim of treatment is to reduce exacerbation rates and slow the decline of the disease. The effect of ICS on mortality is uncertain, and there is some evidence that ICS use may increase rates of pneumonia. Note that asthma can possibly coexist with COPD. Benefits are not seen in patients who continue to smoke.

If corticosteroids are to be used, usual practice is ICS/LABA combination inhalers (fluticasone/salmeterol, fluticasone/vilanterol or budesonide/eformoterol), which are available on the PBS for use in symptomatic patients with moderate to severe COPD ($FEV_1 < 50\%$ predicted).⁹

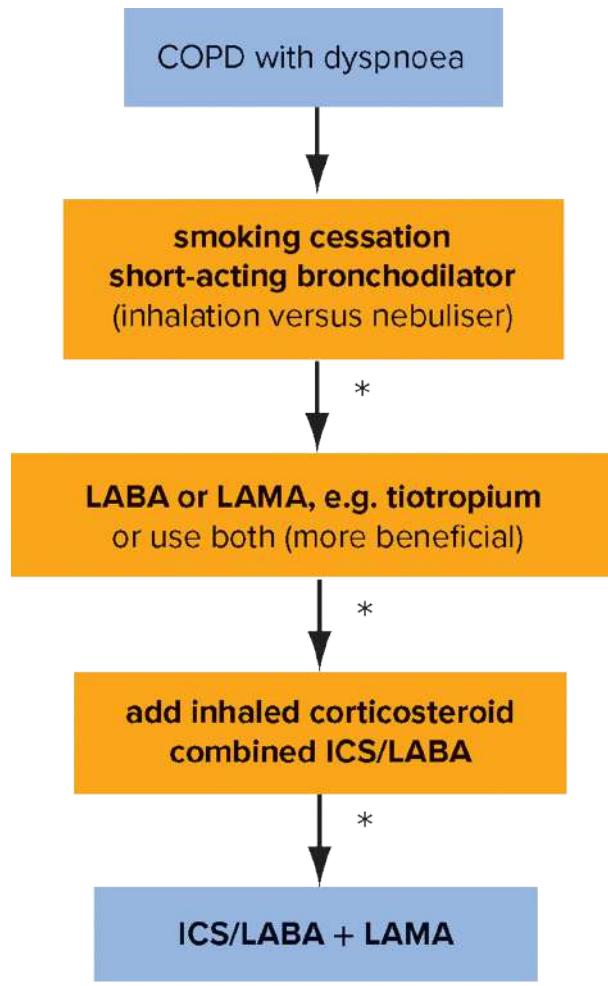
In this group, tiotropium combined with an ICS/LABA combination inhaler (triple therapy) is more beneficial than the individual treatments alone.⁹ Combining LAMA and LABA provides a modest additive effect.¹⁰

Inhaled corticosteroids

Guidelines for prescription include:

- documented but limited evidence of responsiveness to inhaled corticosteroids, including functional status
- those with an $FEV_1 \leq 50\%$ predicted
- two or more exacerbations requiring oral steroids in 12 months

There is no role for ICS monotherapy in the treatment of COPD. Oral corticosteroids are not recommended for maintenance therapy in COPD, although they may be needed in patients with severe COPD where corticosteroids cannot be withdrawn following an acute exacerbation. A stepwise approach to management is outlined in FIGURE 74.2 .



*Inadequate control

FIGURE 74.2 Stepwise approach to management of COPD

P—Prevent deterioration⁵

Reducing risk factors for COPD is a priority, and smoking is the most important and prime target if this continues to be a problem. Stopping is the only measure that slows the progression of COPD. Reinforce patient education programs, and avoid exposure to passive smoke as far as possible.

Annual influenza vaccination

Influenza vaccination reduces the risk of exacerbations, hospitalisation and death. It should be given in early autumn to all patients with COPD.

Pneumococcal vaccination

Vaccination (23vPPV) to prevent invasive bacteraemic pneumococcal pneumonia is recommended, with a booster at 5 years.¹¹

Long-term oxygen therapy

Long-term oxygen therapy (LTOT) reduces mortality in COPD. Long-term continuous Page 885 therapy given for at least 15 hours a day (as close as possible to 24 hours a day) prolongs life in hypoxaemic patients—those who have PaO₂ consistently <55 mmHg (7.3 KPa; SpO₂ 88%) when breathing air. At assessment for ongoing therapy, the patient's condition must be stable and they must have stopped smoking at least 1 month previously. Flow should be set at the lowest rate needed to maintain a resting PaO₂ of 60 mmHg. A flow rate of 0.5–2.0 L/min is usually sufficient. Best used for at least 18 hours a day.⁶ There is no clear-cut evidence about the effectiveness of intermittent ambulatory domiciliary oxygen therapy, but patients with hypoxaemia during sleep may require nocturnal oxygen therapy.

Check current smoking status

Smoking cessation clearly reduces the rate of decline of lung function. GPs and pharmacists can help smokers quit. Brief counselling is effective and every smoker should be offered at least this intervention at every visit.

Refer to strategies in CHAPTER 12 , including effectiveness of treatment for nicotine dependence.

Antibiotics

Current evidence does not support long-term antibiotic use to prevent exacerbations, but they should be used in exacerbations with an increase in cough, dyspnoea, sputum volume or purulence.

Corticosteroids

No medication has yet been shown to prevent the long-term decline in lung function. Inhaled corticosteroids (ICS) are indicated for patients with a documented response or those who have moderate to severe COPD with frequent exacerbations. ICS use has been observed to be associated with increased rates of pneumonia.

Note: Fixed-dose combinations of LABA and ICS (see CHAPTER 73) are often used for patient convenience.⁶

Mucolytic agents

Mucolytic agents may reduce the frequency and duration of exacerbations (evidence level I). Mucolytic therapy should be considered for patients with a chronic cough productive of sputum. Oral mucolytics include potassium iodide, bromhexine, N-acetylcysteine, ambroxol and glyceryl guaiacolate. Compounds containing codeine should be avoided.

Antitussive agents

Regular use of antitussives in stable COPD is contraindicated.

Regular review

Regular review with objective measures of function is recommended in anticipation of reducing complications, frequency and severity of exacerbations and admissions to hospital. Regular schedules for follow-up visits are appropriate. Medication review should involve consideration of deprescribing as well as prescribing.

Lung surgery¹²

The options are bullectomy, lung volume reduction surgery and transplantation. Individuals should be referred for consideration for bullectomy if they have a single large bulla on CT scan associated with breathlessness and an FEV₁ <50% predicted. Other lung surgery should be considered in those with severe COPD who remain breathless with marked restriction of their activities of daily living, despite maximal therapy. Emphysema mainly involving the upper lobes with PaCO₂ <55 mmHg and FEV₁ >20% predicted are some factors required for lung volume reduction surgery.

D—Develop support network and self-management plan⁵

COPD imposes a considerable handicap on patients and carers, with heavy psychosocial issues including fears about the outcome of the disease.

Respiratory physician referral

Early referral to a respiratory physician is appropriate in order to clarify the diagnosis, consider other therapies, consider long-term home oxygen and facilitate organisation of pulmonary rehabilitation.

Pulmonary rehabilitation

One highly effective strategy is pulmonary rehabilitation, which aims to increase patient and carer knowledge and understanding, reduce carer strain and develop positive attitudes towards self-management and exercise. Integrated programs include education, exercise, behaviour modification and support, which are more effective than any separate component.

The support team and multidisciplinary care plans

As the patient's primary health care provider, the GP is uniquely placed to identify smokers and help them quit, facilitate early diagnosis and coordinate the support team. The support team can enhance the quality of life and reduce morbidity for the COPD patient. The support team can include a nurse/respiratory educator, physiotherapist, occupational therapist, social worker, clinical psychologist, speech pathologist, pharmacist and dietitian.

Government and community support services such as Home Care, home maintenance, exercise programs, Meals on Wheels and support groups can be galvanised to provide support. Page 886

Self-management plans

Individuals should be encouraged to take appropriate responsibility for their own management. The primary care team, supported by Chronic Disease Management (formerly known as Extended Primary Care) item numbers, should develop systems to identify those with more severe COPD who might benefit from more focused education and training in self-management skills.

Psychological issues

The issues facing the COPD patient include fear, stress, sleep disturbance, anxiety, panic and depression. Proactive management, including optimal care of these problems as they arise, will facilitate coping. Management also focuses on symptom control and maximising the quality of life. These patients have to face palliative care at the end stage and ethical issues have to be handled sensitively.

Referral to inpatient care

The management plan should include the identification of clinical markers indicating more intensive hospital treatment.

Indications for hospitalisation include:⁵

- rapid rate of onset of acute exacerbation with increased dyspnoea, cough or sputum
- inability to cope at home
- inability to walk between rooms when previously mobile
- severe breathlessness leading to inability to eat or sleep
- inadequate response to ambulatory treatment
- altered mental status suggestive of hypercapnia
- significant comorbidity (e.g. cardiac disease)
- new arrhythmia

- cyanosis

X—manage exacerbations⁵

Diagnosis of an exacerbation is symptomatically the acute onset over minutes to hours of:

- increasing dyspnoea including use of accessory muscles at rest
- increased sputum
- more purulent sputum

Fever may be present, but fever and chest pain are uncommon symptoms. One-third have no identifiable cause, but infections and heavy pollutants can cause the exacerbation. Consider investigating with pulse oximetry, chest X-ray and sputum culture.

Patients should be treated with a bronchodilator, preferably with a large volume spacer. If nebulisers are used they should be driven by compressed air (to avoid the problem of potentially adverse oxygenation). Systemic glucocorticoids reduce the severity and shorten recovery.

Ventilatory support may be used if hypercapnia develops or worsens despite optimal drug therapy. Non-invasive ventilatory support may avoid the need for intubation.

Treatment in summary^{6,13}

Bronchodilators

Initial therapy by SABA inhalation:

salbutamol 100 mcg MDI, up to 8 to 10 inhalations, repeat as required

or

terbutaline 500 mcg DPI, 1 to 2 inhalations, repeat as required

or

ipratropium bromide 20 mcg MDI, up to 4–6 inhalations, repeat as required

If control is inadequate, combine salbutamol or terbutaline with ipratropium bromide.

If a nebuliser is used (which is usually the case upon hospitalisation), use salbutamol 2.5–5 mg, terbutaline 2.5–5 mg or ipratropium bromide 250–500 mcg, as required.

Oxygen therapy⁶

Controlled oxygen delivery—28% via Venturi mask or 2 L/min via nasal prongs should be

commenced if the patient is hypoxaemic (oxygen saturation <92% with pulse oximetry). Maintain the arterial oxyhaemoglobin saturation at 90%. It is important to obtain a direct measurement of arterial blood gases to confirm the degree of hypoxaemia and if hypercapnia or acidosis is present.

Note that those with severe COPD are prone to hypercapnia if they breathe high oxygen concentrations. Supplemental oxygen should be kept to a minimum. If hypercapnia develops, assisted ventilation may be required.

Corticosteroids

Corticosteroids should be used routinely for severe exacerbations. Use:

prednisolone or prednisone 30–50 mg (o), daily

If oral medication cannot be tolerated, use:

Page 887

hydrocortisone 100 mg IV 6 hourly (or equivalent dose of alternative corticosteroid)

Conversion from IV to oral corticosteroid should occur as soon as practicable.

Recent RCTs have clarified that a shorter duration of oral corticosteroid (5 days) is as effective as the traditional 7- to 14-day course for treating COPD exacerbations.^{14,15} The shorter course reduces the known risks of oral corticosteroids, particularly for those individuals who have multiple exacerbations each year. Therefore, the initial prescription should be a 5-day course with the option of a review.

Antibiotics⁶

The use of antibiotics for exacerbations is not routinely indicated as many episodes are due to viral infections. Some patients have repeated exacerbations due to bacterial infection (usually *Haemophilus influenzae*, *S. pneumoniae* or *Moraxella catarrhalis*) where antibiotics have been proven to be beneficial, reducing the risk of mortality by 77%.¹⁶

The indication for antibiotic treatment is:

- increased cough and dyspnoea, *together with*
- increased sputum volume and/or purulence

When indicated, use:

amoxicillin 500 mg (o) 8 hourly for 5 days

or

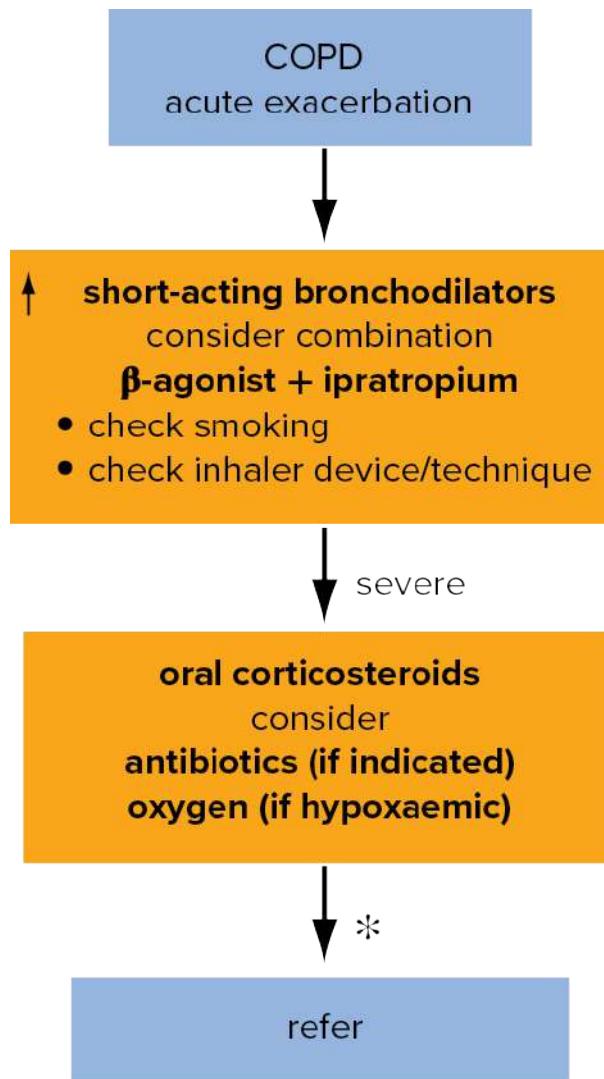
doxycycline 200 mg (o) as 1 dose on day 1, then

100 mg (o) daily for a further 5 days

or

azithromycin 500 mg (o) daily for 5 days

A stepwise approach is summarised in [FIGURE 74.3](#) .



*Inadequate control

FIGURE 74.3 Management plan for acute exacerbation of COPD

Evidence update ^{17,18}

Evidence indicates that regular treatment with long-acting β_2 -agonists is more effective than

treatment with short-acting agents (evidence level I) and is associated with improved quality of life (evidence level II), although they are more costly and do not significantly improve lung function. Current recommended guidelines based on the severity of disease are summarised in TABLE 74.3 .

Table 74.3 COPD therapy according to severity of disease^{9,17}

Stage of COPD	Treatment
0 At risk	Avoidance of risk factors, esp. smoking Influenza and pneumococcal vaccination. ? <i>Haemophilus influenzae</i> vaccination
1 Mild	Add short-acting bronchodilator
2 Moderate	Add long-acting bronchodilators LAMA + LABA Consider LABA/ICS and referral Add pulmonary rehabilitation
3 Severe	Add inhaled corticosteroids LABA/ICS + LAMA
4 Very severe	Add long-term oxygen (if chronic respiratory failure) Consider theophylline (o) or roflumilast Consider surgical referral

Page 888

Practice tips

- The key triad of treatment is: inhaled bronchodilators, smoking cessation and exercise.
- The two priorities are improving lung function and reducing exacerbations. LAMA is a cornerstone of COPD treatment.
- COPD patients should be referred early for rehabilitation. Contact your respiratory physician or hospital for help.
- Pulmonary rehabilitation programs benefit most patients with pulmonary disease.
- Rehabilitation teams are interdisciplinary, usually comprising a rehabilitation physician, physiotherapist, occupational therapist, social worker and dietitian.
- Patients with COPD commonly present in the fifth decade with productive cough

or an acute chest illness.⁴

- Diagnosis can only be established by objective measurement using spirometry with FEV₁ being the preferred parameter.
- Non-invasive positive pressure ventilation (NPPV) reduces mortality and hospital stay in patients with acute failure; it is also an effective weaning strategy for patients who require intubation.¹⁶
- The only treatment proven to slow the progression of COPD is smoking cessation.
- It is very difficult at times to distinguish COPD from the persistent airflow limitation of chronic asthma in older patients (see TABLE 38.3) and the two conditions overlap.
- COPD is reportedly ‘massively’ underdiagnosed—screening with good quality spirometry is important.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Chronic obstructive pulmonary disease
- Bronchitis: chronic bronchitis

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75 Cardiovascular disease

Of all the ailments which may blow out life's little candle, heart disease is the chief.

WILLIAM BOYD (1885–1979), *PATHOLOGY FOR THE SURGEON*

Cardiovascular disease includes mainly:

- coronary heart disease—myocardial ischaemia ([CHAPTER 30](#))
- cerebrovascular disease—strokes and transient ischaemia ([CHAPTER 121](#))
- peripheral vascular disease ([CHAPTER 55](#))

The number one cause of death in the world is coronary heart disease (CHD),¹ whether from sudden fatal acute coronary events, particularly myocardial infarction ([CHAPTER 30](#)) or blocked arteries causing angina and eventually cardiac failure ([CHAPTER 76](#)).

Cardiovascular disease was responsible for 18% of all deaths in Australia in 2017.² This percentage has steadily declined over the past 50 years; in 1968, CVD caused 45% of all deaths and the rate of deaths due to myocardial infarction was around 10 times that of today. This reduction is due to public health measures (particularly decreased smoking rates), improved preventative care (e.g. antihypertensives, statins) and improved emergency medical treatment of acute events. Continuing emphasis on behavioural modification of risk factors and healthy habits is essential to continue this trend.

Risk factors for CVD

Modifiable:

- Hypertension ([CHAPTER 77](#))
- Dyslipidaemia ([CHAPTER 78](#))
- Smoking ([CHAPTER 12](#))
- Diabetes ([CHAPTER 11](#)) with microalbuminuria

- Obesity ([CHAPTER 80](#))
- Sedentary lifestyle
- Alcohol excess ([CHAPTER 12](#))
- Poor nutrition ([CHAPTER 5](#))
- Mental stress³ ([CHAPTER 70](#))

Non-modifiable

- Family history
- Increasing age
- Male gender
- Social history, incl. cultural/ethnic identity

Related conditions:

- Chronic kidney disease ([CHAPTER 79](#)) with microalbuminuria

Assessment of absolute cardiovascular risk

The risk of having a cardiovascular event over the next 5 years (or sometimes 10) is an important assessment which should be estimated before deciding on preventative medication as it greatly influences the pros and cons of screening investigations and treatment. The following target groups should be reassessed every 2 years:

- all adults ≥ 45 years without known history of CVD
- Aboriginal and Torres Strait Islander people ≥ 35 years

People for whom a high CVD risk can be assumed (e.g. established CVD, diabetes in aged >60 , hypertension) do not need an absolute CVD risk assessment using the Framingham Risk Equation, as they automatically fall into the high-risk category.⁴

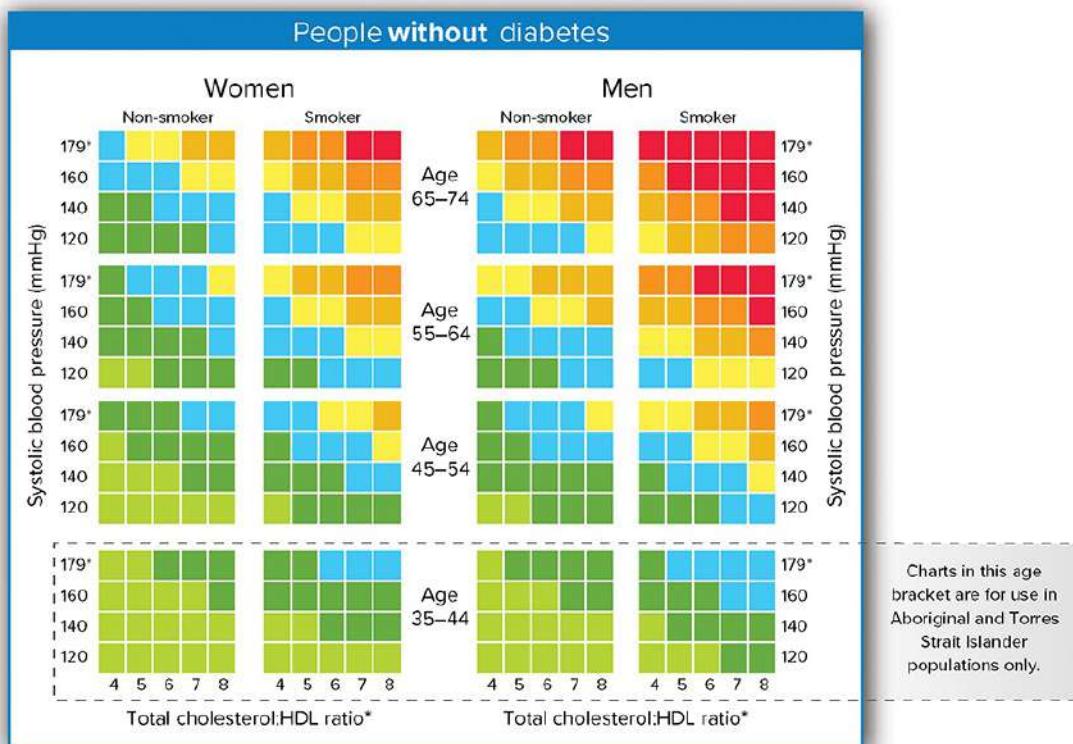
Specific screening recommendations⁴

- Blood pressure should be measured in all adults from age 18 years at least every 2 years.
- Adults should have their fasting blood lipids assessed starting at age 45 years, every 5 years (Aboriginal and Torres Strait Islander people from 35 years). Non-fasting lipids are an acceptable alternative where practicality is an issue.

- Adults should be screened for diabetes (fasting plasma glucose or HbA1c) every 3 years from age 40 years (Aboriginal and Torres Strait Islander people from 18 years).
- Adults at high risk should be screened for kidney disease every 1–2 years (ACR ratio and eGFR).

Estimation of cardiovascular risk guidelines based on the key parameters—Page 890 hypertension, diabetes, smoking, total cholesterol:HDL ratio, age and sex—are presented in [FIGURES 75.1](#) and [75.2](#). Note that BMI, ethnicity and sedentary lifestyle do not form part of the estimates below (or in many other cardiovascular risk tools) even though in practice they may independently influence risk.

Australian cardiovascular risk charts



*In accordance with Australian guidelines, patients with systolic blood pressure ≥ 180 mmHg, or a total cholesterol of >7.5 mmol/L, should be considered at clinically determined high absolute risk of CVD.

Risk level for 5-year cardiovascular (CVD) risk

High risk	Moderate risk	Low risk
≥30%	10–15%	5–9%
25–29%		<5%
20–24%		
16–19%		

How to use the risk charts

- Identify the chart relating to the person's sex, diabetes status, smoking history and age. The charts should be used for all adults aged 45 years or over (and all Aboriginal and Torres Strait Islander adults aged 35–74 years) without known history of CVD and not already known to be at clinically determined high risk.
- Within the chart choose the cell nearest to the person's age, systolic blood pressure (SBP) and cholesterol (TC):HDL ratio. For example, the low cell contains all non-smokers without diabetes who are 34–44 years old and have a TC:HDL ratio of less than 4.5 and a SBP of less than 130 mmHg.
- The colour of the cell that the person falls into provides their 5-year absolute cardiovascular risk level (see legend above for risk category). People fall exactly on a threshold between cells are placed in the cell indicating higher risk.

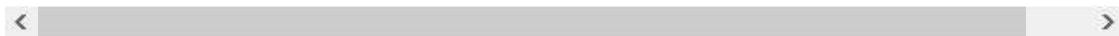
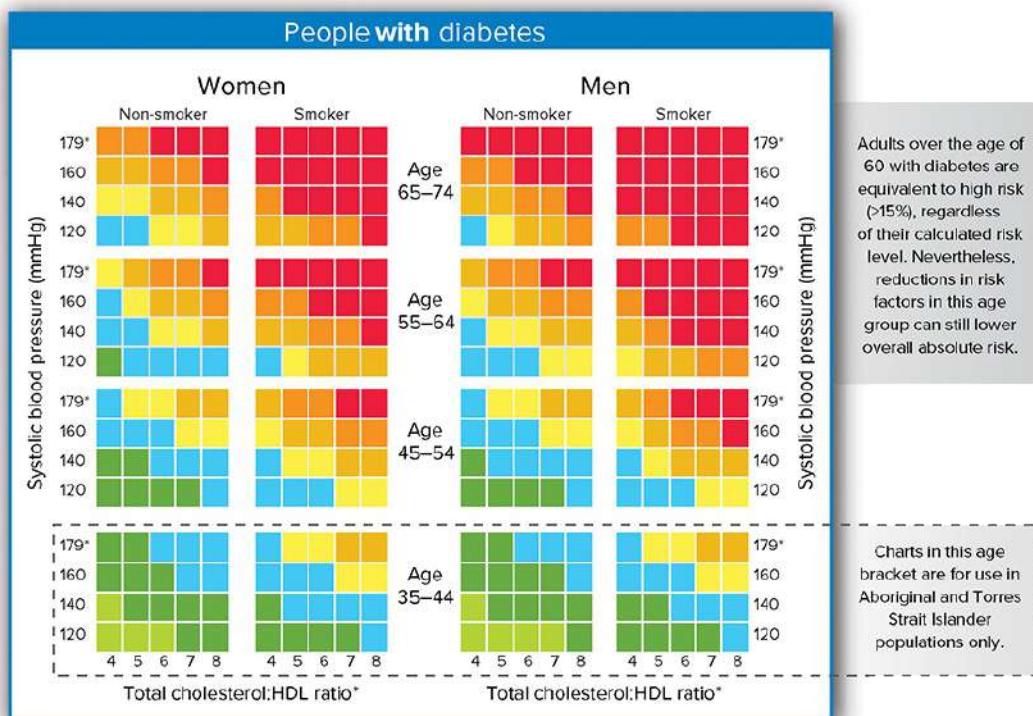


FIGURE 75.1 Estimation of cardiovascular risk in people without diabetes

Source: Used with permission of the National Vascular Disease Prevention Alliance. Absolute Cardiovascular Disease

Australian cardiovascular risk charts



*In accordance with Australian guidelines, patients with systolic blood pressure ≥ 180 mmHg, or a total cholesterol of >7.5 mmol/L, should be considered at clinically determined high absolute risk of CVD.

Risk level for 5-year cardiovascular (CVD) risk



Notes: The risk charts include values for SBP alone as this is the most informative of conventionally measured blood pressure parameters for cardiovascular risk.

For specific groups, additional guidance includes:

The Framingham Risk Equation has not been validated for all population groups, and the assessment score should be interpreted with caution in the following groups:

- The Framingham Risk Equation may **underestimate CVD risk** in Aboriginal and Torres Strait Islander peoples (EBR Grade D); adults with diabetes aged between 45 and 60 years (EBR Grade C); adults aged over 74 years (CBR); however, available evidence suggests that this approach will provide an estimate of minimum cardiovascular risk.

- The Framingham Risk Equation is likely to **underestimate CVD risk** in adults with socioeconomic deprivation (an independent risk factor for cardiovascular disease) (PP) or depression (PP).
- The predictive value of the Framingham Risk Equation **has not been specifically assessed** in adults who are overweight or obese (EBR Grade D).
- The **increased risk of cardiovascular events and all-cause mortality**, in addition to thromboembolic disease including stroke, should be taken into account for adults with atrial fibrillation (particularly those aged over 65 years) (PP).

FIGURE 75.2 Estimation of cardiovascular risk in people with diabetes

Source: Used with permission of the National Vascular Disease Prevention Alliance. Absolute Cardiovascular Disease Management. Quick Reference Guide for health professionals. 2021.

Secondary prevention of CHD for established risk^{5,6}

1 Management of lifestyle/behavioural risk factors with goals

Page 892

All patients who have risk factors should be given lifestyle advice.

Intervention using the 5A framework ([CHAPTER 12](#)) is an appropriate practical approach for general practitioners to assist and encourage patients with behavioural risk factor issues to modify⁷ their behaviour, and can be applied to the following:

- smoking—completely stop smoking and avoid second-hand smoke. The death rate from CHD for smokers is about twice that for non-smokers. Smoking cessation, if applicable, is a priority in risk factor reduction.
- alcohol—consume a low-risk quantity (max. 2 standard drinks per day; 1 standard drink for women with hypertension). See guidelines in [CHAPTER 12](#).
- nutrition—maintain healthy eating including limiting saturated fatty acid and trans fat and salt intake to ≤ 4 g/day.⁷ Aim for a low carbohydrate, healthy fat Mediterranean-type diet. Arrange access to the Heart Foundation Health Information Services and/or refer to a dietitian.
- physical activity—if possible, at least 30 minutes of moderate-intensity physical activity on most days of the week (min. 150 minutes/week). If feasible, arrange an appointment with an accredited exercise physiologist as well as a local activity program.
- healthy weight—waist measurement (men <94 cm, women <80 cm; BMI range 18.5–25). The benefits of weight reduction include improved insulin resistance, decreased LDL-C and triglycerides, and increased HDL-C. See obesity guidelines in [CHAPTER 80](#).
- stress management—encourage promotion of recreation, relaxation techniques, meditation and social support. Treat anxiety disorders and depression with counselling if appropriate.

Additionally, patients at high risk ($>15\%$ 5-year CV risk or previous CV event) should have specific parameters treated.

2 Management of biomedical risk factors with targets

- Blood pressure: $<130/80$ mmHg (or as per hypertension guidelines)
- Lipids: total C <4 mmol/L; LDL-C <2 mmol/L; HDL-C ≥ 1.0 mmol/L; non-HDL-C <2.5 mmol/L; TG <2.0 mmol/L⁵

- Lowering LDL-C is essential to reduce cardiovascular risk; every decrease of 1 mmol/L reduces all-cause mortality by 10%⁸
- Diabetes: maintain optimal blood sugar level 4.0–6.0 mmol/L (ideal); compare NHMRC 6.1–8.0 mmol/L; HbA1c ≤7% (or as per diabetes guidelines)
- Antiplatelet agent: patients with a previous CV event should take aspirin 75–150 mg/day unless contraindicated. However, aspirin is of limited benefit and not routinely recommended in those who have not had a CV event, even if at high risk (>15% 5-year-risk or diabetes).⁹

Resource

National Vascular Disease Prevention Alliance, Guidelines for the management of absolute cardiovascular disease risk. Available from:

https://www.heartfoundation.org.au/getmedia/4342a70f-4487-496e-bbb0-dae33a47fcb2/Absolute-CVD-Risk-Full-Guidelines_2.pdf, accessed April 2021.

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