

6 Management of acute asthma

6.1 LESSONS FROM STUDIES OF ASTHMA DEATHS AND NEAR FATAL ASTHMA

Confidential enquiries into over 200 asthma deaths in the UK have concluded there are factors associated with the disease, the medical management and the patient's behaviour or psychosocial status which contributed to the death. Most deaths occurred before admission to hospital.²¹³⁻²¹⁷

6.1.1 DISEASE FACTORS

Most patients who died of asthma had chronically severe asthma. In a minority the fatal attack occurred suddenly in a patient with only mild or moderately severe background disease.²¹³⁻²¹⁸

2++

6.1.2 MEDICAL MANAGEMENT

Many of the deaths occurred in patients who had received inadequate treatment with inhaled steroid or steroid tablets and/or inadequate objective monitoring of their asthma. Follow up was inadequate in some and others should have been referred earlier for specialist advice. There was widespread under-use of written management plans. Heavy or increasing use of β_2 agonist therapy was associated with asthma death.^{213-217 219 220}

2++

Deaths have continued to be reported following inappropriate prescription of β -blocker therapy or heavy sedation (see section 4.5.5). A small proportion of patients with asthma were sensitive to non-steroidal anti-inflammatory agents; all asthma patients should be asked about past reactions to these agents.

6.1.3 ADVERSE PSYCHOSOCIAL AND BEHAVIOURAL FACTORS

Behavioural and adverse psychosocial factors were recorded in the majority of patients who died of asthma.²¹³⁻²¹⁷ The most important are shown in Table 3.

Table 3: Patients at risk of developing near fatal or fatal asthma

A COMBINATION OF **SEVERE ASTHMA** RECOGNISED BY ONE OR MORE OF:

- previous near fatal asthma, e.g. previous ventilation or respiratory acidosis
- previous admission for asthma especially if in the last year
- requiring three or more classes of asthma medication
- heavy use of β_2 agonist
- repeated attendances at A&E for asthma care especially if in the last year
- brittle asthma.

AND **ADVERSE BEHAVIOURAL OR PSYCHOSOCIAL FEATURES** RECOGNISED BY ONE OR MORE OF:

- non-compliance with treatment or monitoring
- failure to attend appointments
- self-discharge from hospital
- psychosis, depression, other psychiatric illness or deliberate self-harm
- current or recent major tranquilliser use
- denial
- alcohol or drug abuse
- obesity
- learning difficulties
- employment problems
- income problems
- social isolation
- childhood abuse
- severe domestic, marital or legal stress.

Case control studies support most of these observations.^{221 222} Compared with control patients admitted to hospital with asthma, those who died were significantly more likely to have learning difficulties; psychosis or prescribed antipsychotic drugs; financial or employment problems; repeatedly failed to attend appointments or discharged themselves from hospital; drug or alcohol abuse; obesity; or a previous near fatal attack.

2++

Compared with control patients with asthma in the community, patients who died had more severe disease; more likelihood of a hospital admission or visit to A&E for their asthma in the previous year; more likelihood of a previous near fatal attack; poor medical management; failure to measure pulmonary function; and non-compliance.

B Health care professionals must be aware that patients with severe asthma and one or more adverse psychosocial factors are at risk of death.

Studies comparing near fatal asthma with deaths from asthma have concluded that patients with near fatal asthma have identical adverse factors to those described in Table 3, and that these contribute to the near fatal asthma attack.²²³⁻²²⁵ Compared with patients who die, those with near fatal asthma are significantly younger, are significantly more likely to have had a previous near fatal asthma attack, are less likely to have concurrent medical conditions, are less likely to experience delay in receiving medical care, and more likely to have ready access to acute medical care.

2+

Not all patients with near fatal asthma require intermittent positive pressure ventilation.

For those with near fatal asthma, adults as well as children, it is always wise to involve a close relative when discussing future management.

Patients with brittle asthma should also be identified (see section 6.2.3, table 4).

✓ Keep patients who have had near fatal asthma or brittle asthma under specialist supervision indefinitely.

6.1.4 SEASONAL FACTORS

In the UK there is a peak of asthma deaths in younger people (aged up to 44 years) in July and August and in December and January in older people.^{224 226}

2++

6.1.5 PREDICTION AND PREVENTION OF A SEVERE ASTHMA ATTACK

Most (88-92%) attacks of asthma severe enough to require hospital admission develop relatively slowly over a period of six hours or more. In one study, over 80% of attacks developed over more than 48 hours.²²⁷⁻²³² There should therefore be time for effective action and the potential to reduce the number of attacks requiring hospitalisation. There are many similarities between patients who die from asthma, patients with near fatal asthma and asthmatic controls who are admitted to hospital.

2++

✓ A respiratory specialist should follow up patients admitted with severe asthma for at least one year after the admission.

6.2 ACUTE ASTHMA IN ADULTS

Annexes 1-3 contain algorithms summarising the recommended treatment for patients presenting with acute or uncontrolled asthma in primary care (annex 1), A&E (annex 2), and hospital (annex 3).

6.2.1 RECOGNITION OF ACUTE ASTHMA

Definitions of increasing levels of severity of acute asthma exacerbations are provided in Table 4.^{4 7 11 233-235} Predicted PEF values²³⁶ should be used only if the recent best PEF (within two years) is unknown.

2+
4

6.2.2 SELF-TREATMENT BY PATIENTS DEVELOPING ACUTE OR UNCONTROLLED ASTHMA

Many patients with asthma and all patients with severe asthma should have an agreed written action plan and their own peak flow meter, with regular checks of inhaler technique and compliance. They should know when and how to increase their medication and when to seek medical assistance. Asthma action plans have been shown to decrease hospitalisation for²³⁷ and deaths from²³⁸ asthma (see section 9.1.4)

6.2.3 INITIAL ASSESSMENT

All possible initial contact personnel, e.g. practice receptionists, ambulance call takers, NHS Direct (England & Wales), NHS 24 (Scotland), should be aware that asthma patients complaining of respiratory symptoms may be at risk and should have immediate access to a doctor or trained asthma nurse. The assessments required to determine whether the patient is suffering from an acute attack of asthma, the severity of the attack and the nature of treatment required are detailed in Tables 4 and 5. It may also be helpful to use a systematic recording process. Proformas have proved useful in the A&E setting.²³⁹

Table 4: Levels of severity of acute asthma exacerbations

Near fatal asthma	Raised PaCO ₂ and/or requiring mechanical ventilation with raised inflation pressures ²²³⁻²²⁵
Life threatening asthma	Any one of the following in a patient with severe asthma: <ul style="list-style-type: none"> - PEF < 33% best or predicted - SpO₂ < 92% - PaO₂ < 8 kPa - normal PaCO₂ (4.6 – 6.0 kPa) - silent chest - cyanosis - feeble respiratory effort - bradycardia - dysrhythmia - hypotension - exhaustion - confusion - coma
Acute severe asthma	Any one of: <ul style="list-style-type: none"> - PEF 33-50% best or predicted - respiratory rate ≥25/min - heart rate ≥110/min - inability to complete sentences in one breath
Moderate asthma exacerbation	<ul style="list-style-type: none"> - Increasing symptoms - PEF > 50-75% best or predicted - no features of acute severe asthma
Brittle asthma	<ul style="list-style-type: none"> - Type 1: wide PEF variability (> 40% diurnal variation for > 50% of the time over a period > 150 days) despite intense therapy - Type 2: sudden severe attacks on a background of apparently well controlled asthma

6.2.4 PREVENTION OF ACUTE DETERIORATION

A register of patients at risk may help primary care health professionals to identify patients who are more likely to die from their asthma. A system should be in place to ensure that these patients are contacted if they fail to attend for follow up.

6.2.5 CRITERIA FOR REFERRAL

D Refer to hospital any patients with features of acute severe or life threatening asthma.

Other factors, such as failure to respond to treatment, social circumstances or concomitant disease, may warrant hospital referral.

Table 5: Initial assessment: the role of symptoms, signs and measurements

Clinical features	Clinical features, symptoms and respiratory and cardiovascular signs are helpful in recognising some patients with severe asthma, e.g. severe breathlessness (including too breathless to complete sentences in one breath), tachypnea, tachycardia, silent chest, cyanosis or collapse. ^{4 7 11 233-235} <i>None of these singly or together is specific and their absence does not exclude a severe attack.</i>	2+
PEF or FEV₁	Measurements of airway calibre improve recognition of the degree of severity, the appropriateness or intensity of therapy, and decisions about management in hospital or at home. ^{240 241} PEF or FEV ₁ are both useful and valid measures of airway calibre. PEF is more convenient and cheaper. PEF expressed as a percentage of the patient's previous best value is most useful clinically. PEF as a percentage of predicted gives a rough guide in the absence of a known previous best value. Different peak flow meters give different readings. Where possible the same or similar type of peak flow meter should be used. The Nunn & Gregg nomogram is recommended for use with peak flow meter. ⁵³²	2+
Pulse oximetry	Measurement of oxygen saturation (SpO ₂) with a pulse oximeter is necessary in acute severe asthma to determine the adequacy of oxygen therapy and the need for arterial blood gas (ABG) measurement. The aim of oxygen therapy is to maintain SpO ₂ ≥ 92%.	2+
Blood gases (ABG)	Patients with SpO ₂ < 92% or other features of life threatening asthma require ABG measurement. ^{4 7 11 233 235 243}	2+
Chest x-ray	Chest x-ray is not routinely recommended in patients in the absence of: <ul style="list-style-type: none"> - suspected pneumomediastinum or pneumothorax - suspected consolidation - life threatening asthma - failure to respond to treatment satisfactorily - requirement for ventilation. 	4
Systolic paradox	Systolic paradox (<i>pulsus paradoxus</i>) has been abandoned as an indicator of the severity of an attack for pragmatic reasons. ^{4 7 11 233-235 244}	2+

6.2.6 CRITERIA FOR ADMISSION

B Admit patients with any feature of a life threatening or near fatal attack.^{213-217 224 225}

B Admit patients with any feature of a severe attack persisting after initial treatment.^{213-217 224 225}

C Patients whose peak flow is greater than 75% best or predicted one hour after initial treatment may be discharged from A&E, unless they meet any of the following criteria, when admission may be appropriate:

- still have significant symptoms
- concerns about compliance
- living alone / socially isolated
- psychological problems
- physical disability or learning difficulties
- previous near fatal or brittle asthma
- exacerbation despite adequate dose steroid tablets pre-presentation
- presentation at night
- pregnancy.

Criteria for admission in adults are summarised in annexes 1 and 2.

6.3 TREATMENT OF ACUTE ASTHMA IN ADULTS

6.3.1 OXYGEN

Patients with acute severe asthma are hypoxaemic.²⁴⁵⁻²⁴⁸ This should be corrected urgently using high concentrations of inspired oxygen (usually 40-60%) and a high flow mask such as a Hudson mask. Unlike patients with COPD there is little danger of precipitating hypercapnea with high flow oxygen. Hypercapnea indicates the development of near fatal asthma and the need for emergency specialist/anaesthetic intervention. Oxygen saturations of at least 92% must be achieved.

C Give high flow oxygen to all patients with acute severe asthma.

In view of the theoretical risk of oxygen desaturation while using air driven compressors to nebulise β_2 agonist bronchodilators, oxygen-driven nebulisers are the preferred method of delivery in hospitals, ambulances and primary care.^{4 203 249} (NB: In order to generate the flow rate of 6 l/min required to drive most nebulisers, a high flow regulator must be fitted to the oxygen cylinder). The absence of supplemental oxygen should not prevent nebulised therapy from being administered where appropriate.²⁵⁰

- A**
- In hospital, ambulance and primary care, nebulised β_2 agonist bronchodilators should be driven by oxygen.
 - Outside hospital, high dose β_2 agonist bronchodilators may be delivered via large volume spacers or nebulisers.

C Whilst supplemental oxygen is recommended, its absence should not prevent nebulised therapy being given if indicated.

6.3.2 β_2 AGONIST BRONCHODILATORS

In most cases of acute asthma, inhaled β_2 agonists given in high doses act quickly to relieve bronchospasm with few side-effects.²⁵¹⁻²⁵³ There is no evidence for any difference in efficacy between salbutamol and terbutaline, although rarely patients may express a preference. 1+

2004

In acute asthma without life threatening features, β_2 agonists can be administered by repeated activations of a pMDI via an appropriate large volume spacer (**four to six puffs given one at a time and separately inhaled at intervals of 10-20 minutes**) or by wet nebulisation driven by oxygen, if available.²⁰³ Inhaled β_2 agonists are at least as efficacious and preferable to intravenous β_2 agonists (meta-analysis has excluded subcutaneous trials) in adult acute asthma in the majority of cases.²⁵⁴ 1++

A Use high dose inhaled β_2 agonists as first line agents in acute asthma and administer as early as possible. Intravenous β_2 agonists should be reserved for those patients in whom inhaled therapy cannot be used reliably.

✓ In acute asthma with life threatening features the nebulised route (oxygen-driven) is recommended

Parenteral β_2 agonists, in addition to inhaled β_2 agonists, may have a role in ventilated patients or those patients *in extremis* in whom nebulised therapy may fail; however there is limited evidence to support this.

2004

Continuous nebulisation of β_2 agonists is at least as efficacious as bolus nebulisation in relieving acute asthma. It is more effective in airflow obstruction that is severe or unresponsive to initial treatment.⁵³¹ However, most cases of acute asthma will respond adequately to bolus nebulisation of β_2 agonists. 1+

2004

A In severe asthma (PEF or FEV₁ < 50% best or predicted) and asthma that is poorly responsive to an initial bolus dose of β_2 agonist, **consider continuous nebulisation, using an appropriate nebuliser system.**

2004

Continuous nebulisation cannot be achieved with all nebuliser systems, and is not equivalent to continuously repeating conventional nebuliser doses. 4

Repeated doses of β_2 agonists should be given at 15-30 minute intervals or continuous nebulisation of salbutamol at 5-10 mg/hour (requires appropriate nebuliser) used if there is an inadequate response to initial treatment. Higher bolus doses, e.g. 10 mg of salbutamol, are unlikely to be more effective.

6.3.3 STEROID THERAPY

Steroid tablets reduce mortality, relapses, subsequent hospital admission and requirement for β_2 agonist therapy. The earlier they are given in the acute attack the better the outcome.^{258 259} 1++

A Give steroid tablets in adequate doses in all cases of acute asthma.

Steroid tablets are as effective as injected steroids, provided tablets can be swallowed and retained.²⁵⁸ Doses of prednisolone of 40-50 mg daily or parenteral hydrocortisone 400 mg daily (100 mg six-hourly) are as effective as higher doses.²⁶⁰ For convenience, steroid tablets may be given as 2 x 25 mg tablets daily rather than 8-12 x 5 mg tablets. 1++

✓ Continue prednisolone 40-50 mg daily for at least five days or until recovery.

Following recovery from the acute exacerbation steroid tablets can be stopped abruptly and doses do not need tapering provided the patient receives inhaled steroids^{261 262} (apart from patients on maintenance steroid treatment or rare instances where steroids are required for three or more weeks). 1+

There is no evidence to suggest that inhaled steroids should be substituted for steroid tablets in treating patients with acute severe, or life threatening asthma. Further randomised controlled trials to determine the role of inhaled steroids in these patients are required.

Inhaled steroids do not provide benefit in addition to the initial treatment,²⁶³ but should be continued (or started as soon as possible) to form the start of the chronic asthma management plan. 1++

6.3.4 IPRATROPIUM BROMIDE

Combining nebulised ipratropium bromide with a nebulised β_2 agonist has been shown to produce significantly greater bronchodilation than a β_2 agonist alone, leading to a faster recovery and shorter duration of admission. Anticholinergic treatment is not necessary and may not be beneficial in milder exacerbations of asthma or after stabilisation.²⁶⁴⁻²⁶⁶

1⁺⁺

A Nebulised ipratropium bromide (0.5 mg 4-6 hourly) should be added to β_2 agonist treatment for patients with acute severe or life threatening asthma or those with a poor initial response to β_2 agonist therapy.

6.3.5 INTRAVENOUS MAGNESIUM SULPHATE

2004

A single dose of IV magnesium sulphate has been shown to be safe and effective in acute severe asthma who have not had a good initial response to treatment.²⁶⁷

1⁺⁺

The safety and efficacy of repeated doses have not been assessed in patients with asthma. Repeated doses could give rise to hypermagnesaemia with muscle weakness and respiratory failure.

A Consider giving a single dose of IV magnesium sulphate for patients with:

- acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy
- life threatening or near fatal asthma.

✓ IV magnesium sulphate (1.2-2 g IV infusion over 20 minutes) should only be used following consultation with senior medical staff.

More studies are needed to determine the optimal frequency and dose of IV magnesium sulphate therapy.

6.3.6 INTRAVENOUS AMINOPHYLLINE

In acute asthma, the use of IV aminophylline is not likely to result in any additional bronchodilation compared to standard care with inhaled bronchodilators and steroid tablets. Side-effects such as palpitations, arrhythmias and vomiting are increased if IV aminophylline is used.²⁶⁸

1⁺⁺

✓ Use IV aminophylline only after consultation with senior medical staff.

Some individual patients with near fatal asthma or life threatening asthma with a poor response to initial therapy may gain additional benefit from IV aminophylline (5 mg/kg loading dose over 20 minutes unless on maintenance oral therapy, then infusion of 0.5-0.7 mg/kg/hr). Such patients are probably rare and could not be identified in a meta-analysis of trials involving 739 subjects.²⁶⁸ If IV aminophylline is given to patients on oral aminophylline or theophylline, blood levels should be checked on admission. Levels should be checked daily for all patients on aminophylline infusions.

6.3.7 LEUKOTRIENE RECEPTOR ANTAGONISTS

There is no published study of the use of leukotriene receptor antagonists in the management of acute asthma

6.3.8 ANTIBIOTICS

When an infection precipitates an exacerbation of asthma it is likely to be viral in type. The role of bacterial infection has been overestimated.²⁶⁹

1⁺⁺

B Routine prescription of antibiotics is not indicated for acute asthma.

6.3.9 HELIOX

The use of heliox (helium/oxygen mixture in a ratio of 80:20 or 70:30) in acute adult asthma cannot be recommended on the basis of present evidence.²⁷⁰⁻²⁷¹

1⁺

6.3.10 INTRAVENOUS FLUIDS

There are no controlled trials or even observational or cohort studies of differing fluid regimes in acute asthma. Some patients with acute asthma require rehydration and correction of electrolyte imbalance. Hypokalaemia can be caused or exacerbated by β_2 agonist and/or steroid treatment and must be corrected.

6.3.11 REFERRAL TO INTENSIVE CARE

Indications for admission to intensive care facilities or a high dependency unit include patients requiring ventilatory support and those with severe acute or life threatening asthma who are failing to respond to therapy, as evidenced by:

- deteriorating PEF
- persisting or worsening hypoxia
- hypercapnea
- arterial blood gas analysis showing fall in pH or rising H^+ concentration
- exhaustion, feeble respiration
- drowsiness, confusion
- coma or respiratory arrest.⁴⁷

2⁺

Not all patients admitted to the Intensive Care Unit (ICU) need ventilation, but those with worsening hypoxia or hypercapnea, drowsiness or unconsciousness and those who have had a respiratory arrest require intermittent positive pressure ventilation. Intubation in such patients is very difficult and should ideally be performed by an anaesthetist or ICU consultant.⁴⁷ Intensive care management is outwith the remit of these guidelines.

C All patients transferred to intensive care units should be accompanied by a doctor suitably equipped and skilled to intubate if necessary.

6.3.12 NON-INVASIVE VENTILATION

Non-invasive ventilation (NIV) is now well established in the management of ventilatory failure caused by extrapulmonary restrictive conditions and exacerbations of COPD. Hypercapneic respiratory failure developing during the evolution of an acute asthmatic episode is regarded as an indication for urgent admission to the ICU. It is unlikely that NIV would ever replace intubation in these very unstable patients but it has been suggested that this treatment can be used safely and effectively.²⁷² Future studies might usefully examine its role in the gradually tiring patient, but at present this treatment cannot be recommended outside randomised controlled trials.

4

6.4 FURTHER INVESTIGATION AND MONITORING

- ✓ ■ Measure and record PEF 15-30 minutes after starting treatment, and thereafter according to the response. Measure and record PEF before and after nebulised or inhaled β_2 agonist bronchodilator (at least four times daily) throughout the hospital stay and until controlled after discharge.
- Record oxygen saturation by oximetry and maintain arterial $SaO_2 > 92\%$
- Repeat measurements of blood gas tensions within two hours of starting treatment if:
 - the initial PaO_2 is < 8 kPa unless SaO_2 is $> 92\%$; or
 - the initial $PaCO_2$ is normal or raised; or
 - the patient's condition deteriorates.
- ✓ ■ Measure them again if the patient's condition has not improved by 4-6 hours.
- Measure and record the heart rate.
- Measure serum potassium and blood glucose concentrations.
- Measure the serum theophylline concentration if aminophylline is continued for more than 24 hours (aim at a concentration of 55-110 $\mu\text{mol/l}$).

6.5 ASTHMA MANAGEMENT PROTOCOLS AND PROFORMAS

The use of structured proformas has been shown to facilitate improvements in the process of care in A&E departments and hospital wards and to improve patient outcomes. The use of this type of documentation can assist data collection aimed at determining quality of care and outcomes.^{239 273-275}

2++

6.6 HOSPITAL DISCHARGE AND FOLLOW UP (see annex 3)

6.6.1 TIMING OF DISCHARGE

There is no single physiological parameter that defines absolutely the timing of discharge from an admission with acute asthma. Patients should have clinical signs compatible with home management, be on reducing amounts of β_2 agonist (preferably no more than four hourly) and be on medical therapy they can continue safely at home.

Although diurnal variability of PEF is not always present during an exacerbation, evidence suggests that patients discharged with PEF < 75% best or predicted and with diurnal variability > 25% are at greater risk of early relapse and readmission.^{276 277}

2+

6.6.2 PATIENT EDUCATION

Following discharge from hospital or A&E departments, a proportion of patients re-attend A&E departments, with more than 15% re-attending within two weeks. Some repeat attenders need emergency care, but many delay seeking help, and are under-treated and/or under-monitored.²⁷⁸

2+

Prior to discharge, trained staff should give asthma education. This should include education on inhaler technique and PEF record keeping, with a written PEF and symptom-based action plan being provided allowing the patient to adjust their therapy within recommendations. These measures have been shown to reduce morbidity after the exacerbation and reduce relapse rates.²⁷⁹

1++

There is some experience of a discrete population of patients who inappropriately use A&E departments rather than the primary care services for their asthma care.²⁸⁰

For the above groups there is a role for a trained asthma liaison nurse based in, or associated with, the A&E department.

6.6.3 FOLLOW UP

A careful history should elicit the reasons for the exacerbation and explore possible actions the patient should take to prevent future emergency presentations.

Medication should be altered depending upon the assessment and the patient provided with an asthma action plan aimed at preventing relapse, optimising treatment and preventing delay in seeking assistance in the future.

Follow up should be arranged prior to discharge with the patient's general practitioner or asthma nurse within two working days; and with a hospital specialist asthma nurse or respiratory physician at about one month after admission.

Recommendations for follow up after acute exacerbations of asthma are covered in more detail in section 9.2.

✓ It is essential that the patient's primary care practice is informed within 24 hours of discharge from A&E or hospital following an asthma exacerbation treated in hospital. Ideally this communication should be directly with a named individual responsible for asthma care within the practice, by means of fax or e-mail.

6.7 ACUTE ASTHMA IN CHILDREN AGED OVER 2 YEARS

6.7.1 INITIAL ASSESSMENT

Table 6 details criteria for assessment of severity of acute asthma attacks in children. See also annexes 4-6.

Table 6: Clinical features for assessment of severity

Acute severe	Life threatening
Can't complete sentences in one breath or too breathless to talk or feed	<ul style="list-style-type: none"> ■ Silent chest ■ Cyanosis ■ Poor respiratory effort
Pulse > 120 in children aged > 5 years > 130 in children aged 2-5 years	<ul style="list-style-type: none"> ■ Hypotension ■ Exhaustion
Respiration > 30 breaths/min aged > 5 years > 50 breaths/min aged 2-5 years	<ul style="list-style-type: none"> ■ Confusion ■ Coma

Before children can receive appropriate treatment for acute asthma in any setting, it is essential to assess accurately the severity of their symptoms. The following clinical signs should be recorded:

- Pulse rate
(increasing tachycardia generally denotes worsening asthma; a fall in heart rate in life threatening asthma is a pre-terminal event).
- Respiratory rate and degree of breathlessness
(i.e. too breathless to complete sentences in one breath or to feed).
- Use of accessory muscles of respiration
(best noted by palpation of neck muscles).
- Amount of wheezing
(which might become biphasic or less apparent with increasing airways obstruction).
- Degree of agitation and conscious level
(always give calm reassurance).

Clinical signs correlate poorly with the severity of airways obstruction.²⁸¹⁻²⁸⁴ Some children with acute severe asthma do not appear distressed.

2⁺⁺

Objective measurements of PEF and SpO₂ are essential. Suitable equipment should be available for use by all health professionals assessing acute asthma in both primary and secondary care settings.

Low oxygen saturations after initial bronchodilator treatment selects a more severe group of patients.^{281 284}

2⁺⁺

B Consider intensive inpatient treatment for children with SpO₂ < 92% on air after initial bronchodilator treatment.

- ✓ Decisions about admission should be made by trained physicians after repeated assessment of the response to further bronchodilator treatment.

A measurement of < 50% predicted PEF or FEV₁ with poor improvement after initial bronchodilator treatment is predictive of a more prolonged asthma attack.

- ✓ Attempt to measure PEF or FEV₁ in all children aged > 5 years, taking the best of three measurements, ideally expressed as percentage of personal best for PEF (as detailed in a written action plan) or alternatively as percentage of predicted for PEF or FEV₁.

Chest x-rays and ABG measurements rarely provide additional useful information and are not routinely indicated.^{285 286}

6.8 TREATMENT OF ACUTE ASTHMA IN CHILDREN AGED OVER 2 YEARS

Emergency units attending to children with acute asthma should have a registered sick children's nurse available on duty at all times and staff familiar with the specific needs of children. The use of proformas can increase the accuracy of severity assessment.

An assessment-driven algorithm has been shown to reduce treatment costs and hospital stay.²⁸⁷ | 2+

D The use of structured care protocols detailing bronchodilator usage, clinical assessment, and specific criteria for safe discharge is recommended.

6.8.1 OXYGEN

✓ Children with life threatening asthma or SpO₂ < 92% should receive high flow oxygen via a tight fitting face mask or nasal cannula at sufficient flow rates to achieve normal saturations.

6.8.2 β₂AGONIST BRONCHODILATORS

A Inhaled β₂ agonists are the first line treatment for acute asthma.²⁸⁸⁻²⁹¹

pMDI + spacer is an effective alternative to nebulisers for bronchodilator inhalation to treat mild to moderate asthma.^{203 292} Children receiving β₂ agonists via pMDI + spacer are less likely to have tachycardia and hypoxia than when the same drug is given via a nebuliser.²⁰³ | 1+

A pMDI + spacer are the preferred option in mild to moderate asthma.

Information about implementing evidence-based guidelines using such devices has been published.²⁹³ Children aged < 3 years are likely to require a face mask connected to the mouthpiece of a spacer for successful drug delivery. Inhalers should be actuated into the spacer in individual puffs and inhaled immediately by tidal breathing.

Frequent doses of β₂ agonists are safe for the treatment of acute asthma,²⁸⁸⁻²⁹⁰ although children with mild symptoms benefit from lower doses.²⁹¹ | 1+

B Individualise drug dosing according to severity and adjust according to the patient's response.

Two to four puffs repeated every 20-30 minutes according to clinical response might be sufficient for mild attacks although up to 10 puffs might be needed for more severe asthma.

✓ Children with acute asthma in primary care who have not improved after receiving up to 10 puffs of β₂ agonist should be referred to hospital. Further doses of bronchodilator should be given as necessary whilst awaiting transfer.

✓ Treat children transported to hospital by ambulance with oxygen and nebulised β₂ agonists during the journey.

✓ Transfer children with severe or life threatening asthma urgently to hospital to receive frequent doses of nebulised β₂ agonists (2.5-5 mg salbutamol or 5-10 mg terbutaline).

Doses can be repeated every 20-30 minutes. Continuous nebulised β₂ agonists are of no greater benefit than the use of frequent intermittent doses in the same total hourly dosage.^{294 295}

6.8.3 IV SALBUTAMOL

The role of intravenous β₂ agonists in addition to nebulised treatment remains unclear.²⁵⁴ One study has shown that an IV bolus of salbutamol given in addition to near maximal doses of nebulised salbutamol results in clinically significant benefits.²⁵⁴ | 1+

B The early addition of a bolus dose of intravenous salbutamol (15 mcg/kg) can be an effective adjunct to treatment in severe cases.

Continuous intravenous infusion should be considered when there is uncertainty about reliable inhalation or for severe refractory asthma. Doses above 1-2 mcg/kg/min (200 mcg/ml solution) should be given in a Paediatric Intensive Care Unit (PICU) setting (up to 5 mcg/kg/min) with regular monitoring of electrolytes.

6.8.4 STEROID THERAPY

Steroid tablets

The early use of steroids for acute asthma can reduce the need for hospital admission and prevent a relapse in symptoms after initial presentation.^{258 259} Benefits can be apparent within three to four hours.

2+

1+

A Give prednisolone early in the treatment of acute asthma attacks.

A soluble preparation dissolved in a spoonful of water is preferable in those unable to swallow tablets. Use a dose of 20 mg for children 2-5 years old and 30-40 mg for children > 5 years.

Oral and intravenous steroids are of similar efficacy.^{260 296 297} Intravenous hydrocortisone (4 mg/kg repeated four hourly) should be reserved for severely affected children who are unable to retain oral medication.

1+

Larger doses do not appear to offer a therapeutic advantage for the majority of children.²⁹⁸ There is no need to taper the dose of steroid tablets at the end of treatment.

2+

- ✓ ■ Use a dose of 20 mg prednisolone for children aged 2-5 years and a dose of 30-40 mg for children > 5 years. Those already receiving maintenance steroid tablets should receive 2 mg/kg prednisolone up to a maximum dose of 60 mg.
- Repeat the dose of prednisolone in children who vomit and consider intravenous steroids in those who are unable to retain orally ingested medication.
- Treatment for up to three days is usually sufficient, but the length of course should be tailored to the number of days necessary to bring about recovery.

Inhaled steroids

There is insufficient evidence to support the use of inhaled steroids as alternative or additional treatment to steroid tablets for acute asthma.^{263 299-301}

- ✓ Do not initiate inhaled steroids in preference to steroid tablets to treat acute childhood asthma.

Children with chronic asthma not receiving regular preventive treatment will benefit from initiating inhaled steroids as part of their long term management. There is no evidence that increasing the dose of inhaled steroids is effective in treating acute symptoms, but it is good practice for children already receiving inhaled steroids to continue with their usual maintenance doses.

6.8.5 IPRATROPIUM BROMIDE

There is good evidence for the safety and efficacy of frequent doses of ipratropium bromide used in addition to β_2 agonists for the first two hours of a severe asthma attack. Benefits are more apparent in the most severe patients.³⁰²

1+

A If symptoms are refractory to initial β_2 agonist treatment, add ipratropium bromide (250 mcg/dose mixed with the nebulised β_2 agonist solution).

Frequent doses up to every 20-30 minutes (250 mcg/dose mixed with the β_2 agonist solution in the same nebuliser) should be used early. The dose frequency should be reduced as clinical improvement occurs.

- ✓ Repeated doses of ipratropium bromide should be given early to treat children poorly responsive to β_2 agonists.

Children with continuing severe asthma despite frequent nebulised β_2 agonists and ipratropium bromide and those with life threatening features need urgent review by a specialist with a view to transfer to a High Dependency Unit or PICU.

6.8.6 IV AMINOPHYLLINE

There is no evidence that aminophylline is of benefit for mild to moderate asthma and side-effects are common and troublesome.^{268 303} However, one well conducted study has shown evidence for benefit in severe acute asthma unresponsive to multiple doses of β_2 agonists and steroids.³⁰⁴ | 1+
2+

A Aminophylline is not recommended in children with mild to moderate acute asthma.

C Consider aminophylline in a High Dependency Unit or PICU setting for children with severe or life threatening bronchospasm unresponsive to maximal doses of bronchodilators and steroid tablets.

A 5 mg/kg loading dose should be given over 20 minutes with ECG monitoring (omit in those receiving maintenance oral theophyllines) followed by a continuous infusion at 1 mg/kg/hour. Estimate serum theophylline levels in patients already receiving oral treatment and in those receiving prolonged treatment.

6.8.7 OTHER THERAPIES

There is no evidence to support the use of heliox or leukotriene receptor antagonists for the treatment of acute asthma in childhood.

There is insufficient evidence to support or refute the role of antibiotics in acute asthma,³⁰⁵ but the majority of acute asthma attacks are triggered by viral infection.

✓ Do not give antibiotics routinely in the management of acute childhood asthma.

6.8.8 INTRAVENOUS FLUIDS

Children with prolonged severe asthma not tolerating oral fluids will require intravenous hydration. Two thirds of the child's maintenance requirement should be given because of the possibility of inappropriate antidiuretic hormone secretion. Serum electrolytes should be measured and hypokalaemia corrected if detected.

✓ ECG monitoring is mandatory for all intravenous treatments.

6.8.9 IV MAGNESIUM SULPHATE

Intravenous magnesium sulphate is a safe treatment for acute asthma although its place in management is not yet established.^{267 306} Doses of up to 40 mg/kg/day (maximum 2 g) by slow infusion have been used. Studies of efficacy for severe childhood asthma unresponsive to more conventional therapies have been inconsistent in providing evidence of benefit. | 1+

6.8.10 FURTHER INVESTIGATION AND MONITORING

Children can be discharged when stable on 3-4 hourly inhaled bronchodilators that can be continued at home.³⁰⁷ PEF and/or FEV₁ should be >75% of best or predicted and SpO₂ >94%.

Adult studies show that "optimal care" comprising self-monitoring, regular review and a written asthma action plan can improve outcomes.²³⁷ Acute asthma attacks should be considered a failure of preventive therapy and thought should be given about how to help families avoid further severe episodes. Discharge plans should address the following:

- check inhaler technique
- consider the need for regular inhaled steroids
- provide a written asthma action plan for subsequent asthma with clear instructions about the use of bronchodilators, seeking urgent medical attention in the event of worsening symptoms and, if appropriate, starting a course of oral steroids
- arrange follow up by a GP within one week
- arrange follow up in a paediatric asthma clinic within one to two months.

6.9 ASSESSMENT OF ACUTE ASTHMA IN CHILDREN AGED LESS THAN 2 YEARS (see annex 7)

The assessment of acute asthma in early childhood can be difficult. Intermittent wheezing attacks are usually due to viral infection and the response to asthma medication is inconsistent. Prematurity and low birth weight are risk factors for recurrent wheezing. The differential diagnosis of symptoms includes aspiration pneumonitis, pneumonia, bronchiolitis, tracheomalacia, and complications of underlying conditions such as congenital anomalies and cystic fibrosis. These guidelines are intended for those who are thought to have asthma causing acute wheeze. They should not be used as a guide for treating acute bronchiolitis. (See forthcoming SIGN guideline on bronchiolitis in children).

6.10 TREATMENT OF ACUTE ASTHMA IN CHILDREN AGED LESS THAN 2 YEARS

6.10.1 β_2 AGONIST BRONCHODILATORS

A trial of bronchodilator therapy should be considered when symptoms are of concern. If inhalers have been successfully administered but there is no response, review the diagnosis and consider the use of other treatment options.

Oral β_2 agonists have not been shown to affect symptom score or length of hospital stay for acute asthma in infancy when compared to placebo.³⁰⁸ | 1+

B Oral β_2 agonists are not recommended for acute asthma in infants.

Inhaled β_2 agonists are the treatment of choice for the initial treatment of acute asthma. Close fitting face masks are essential for optimal drug delivery. The dose received is increased if the child is breathing appropriately and not taking large gasps because of distress and screaming.

There is good evidence that pMDI + spacer is as effective as, if not better than, nebulisers for treating mild to moderate asthma in children aged < 2 years.^{205 309 310} | 1+

A For mild to moderate acute asthma, a pMDI + spacer is the optimal drug delivery device.

Whilst β_2 agonists offer marginal benefits to children aged < 2 years with acute wheeze, there is little evidence for an impact on the need for hospital admission or length of hospital stay.³¹¹⁻³¹³ | 1+

6.10.2 STEROID THERAPY

Steroid tablets in conjunction with β_2 agonists have been shown to reduce hospital admission rates when used in the emergency department.³¹⁴ Steroid tablets have also been shown to reduce the length of hospital stay.^{308 311 314} | 1+

B Consider steroid tablets in infants early in the management of moderate to severe episodes of acute asthma in the hospital setting.

One study has shown similar benefits when comparing oral and nebulised steroids for acute asthma.³¹¹

✓ Steroid tablet therapy (10 mg of soluble prednisolone for up to three days) is the preferred steroid preparation for use in this age group.

6.10.3 IPRATROPIUM BROMIDE

The addition of ipratropium bromide to β_2 agonists for acute severe asthma may lead to some improvement in clinical symptoms and reduce the need for more intensive treatment. It does not reduce the length of hospital stay either in combination with β_2 agonists or in comparison with placebo.³¹⁵ | 1+

B Consider inhaled ipratropium bromide in combination with an inhaled β_2 agonist for more severe symptoms.

6.10.4 FURTHER INVESTIGATION AND MONITORING

Many children with recurrent episodes of viral-induced wheezing in infancy do not go on to have chronic atopic asthma. The majority do not require treatment with regular inhaled steroids. Parents should be advised about the relationship between cigarette smoke exposure and wheezy illnesses (see *sections 3.1 & 3.3*). Referral to suitable agencies should be offered to those who wish to give up smoking.

Parents of wheezy infants should receive appropriate discharge plans along similar lines to those given for older children (see *section 6.8.10*).

Annex 1

Management of acute severe asthma in adults in general practice

Many deaths from asthma are preventable, but delay can be fatal. Factors leading to poor outcome include:

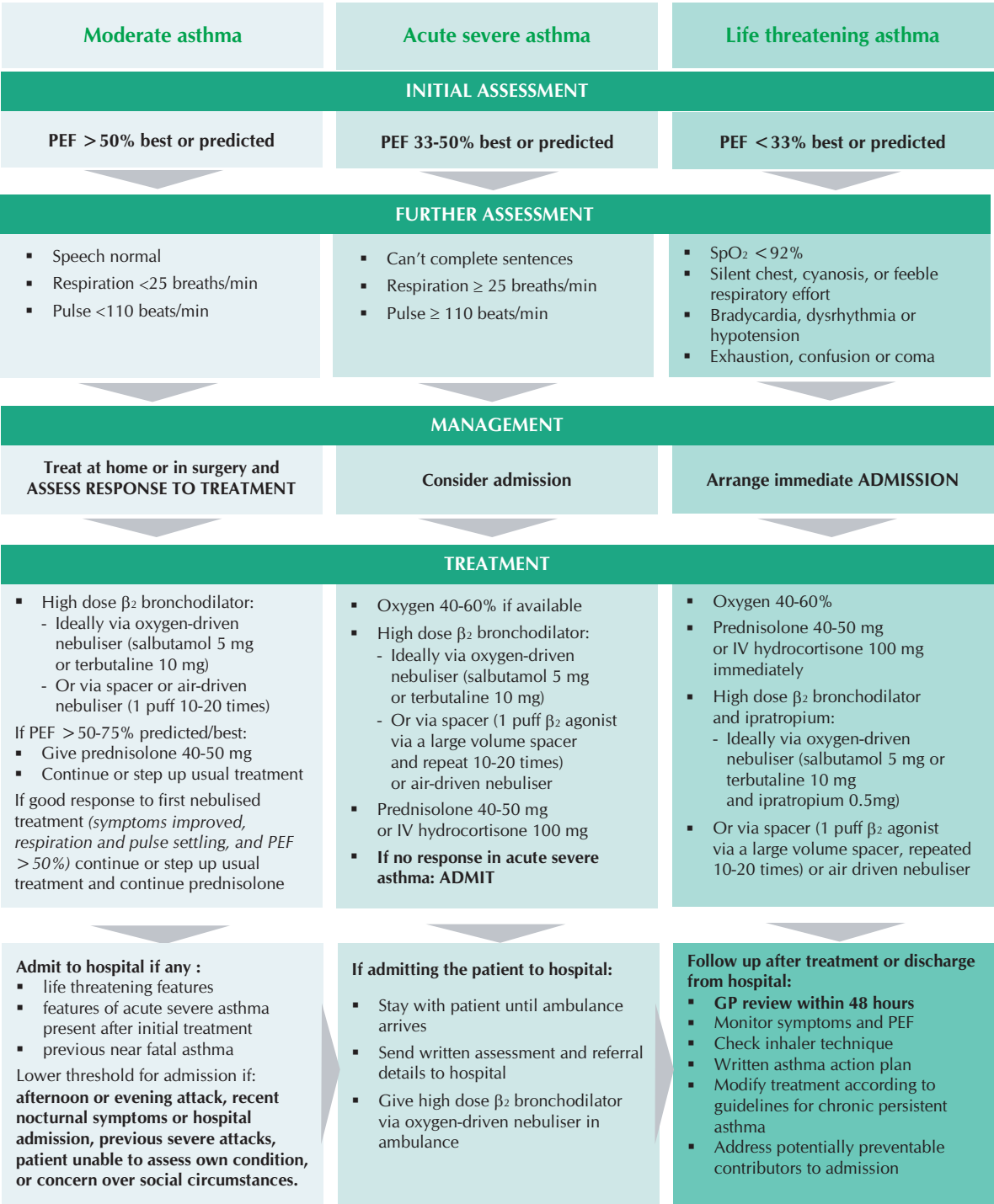
- Doctors failing to assess severity by objective measurement
- Patients or relatives failing to appreciate severity
- Under use of corticosteroids

Regard each emergency asthma consultation as for acute severe asthma until it is shown to be otherwise.

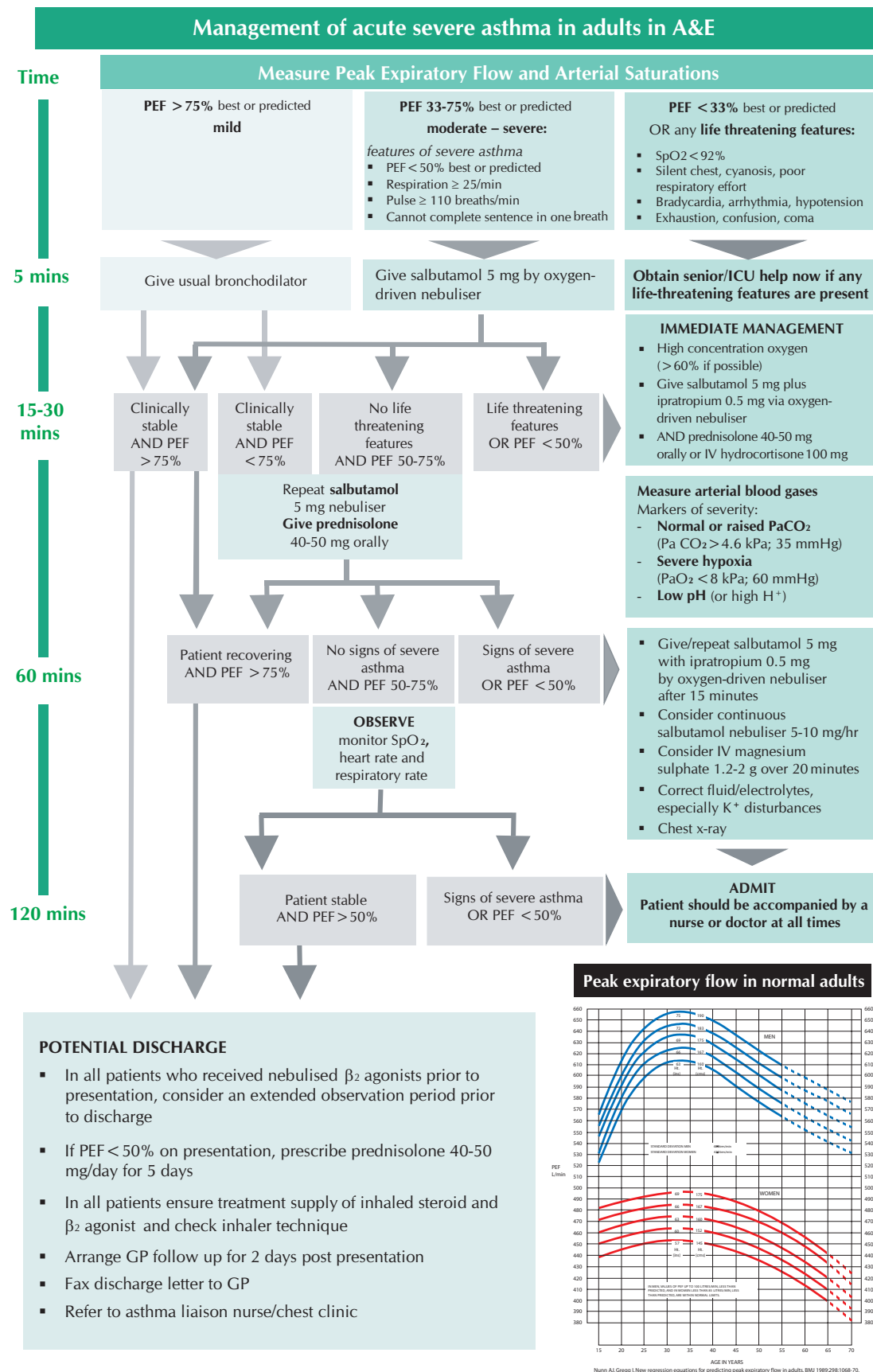
Assess and record:

- Peak expiratory flow (PEF)
- Symptoms and response to self treatment
- Heart and respiratory rates
- Oxygen saturation (by pulse oximetry, if available)

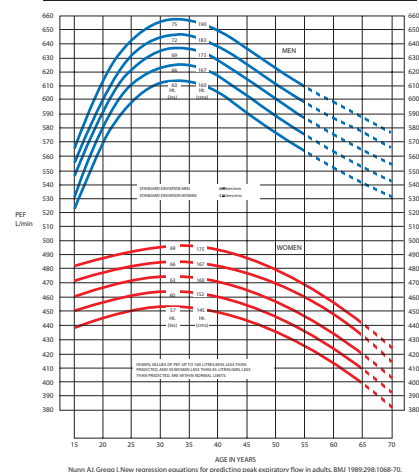
Caution: Patients with severe or life threatening attacks may not be distressed and may not have all the abnormalities listed below. The presence of any should alert the doctor.



Annex 2



Peak expiratory flow in normal adults



Annex 3

Management of acute severe asthma in adults in hospital

Features of acute severe asthma

- Peak expiratory flow (PEF) 33-50% of best (use % predicted if recent best unknown)
- Can't complete sentences in one breath
- Respirations ≥ 25 breaths/min
- Pulse ≥ 110 beats/min

Life threatening features

- PEF $< 33\%$ of best or predicted
- SpO₂ $< 92\%$
- Silent chest, cyanosis, or feeble respiratory effort
- Bradycardia, dysrhythmia, or hypotension
- Exhaustion, confusion, or coma

If a patient has any life threatening feature, measure arterial blood gases. No other investigations are needed for immediate management.

Blood gas markers of a life threatening attack:

- Normal (4.6-6 kPa, 35-45 mmHg) PaCO₂
- Severe hypoxia: PaO₂ < 8 kPa (60mmHg) irrespective of treatment with oxygen
- A low pH (or high H⁺)

Caution: Patients with severe or life threatening attacks may not be distressed and may not have all these abnormalities. The presence of any should alert the doctor.

Near fatal asthma

- Raised PaCO₂
- Requiring IPPV with raised inflation pressures

IMMEDIATE TREATMENT

- Oxygen 40-60% (CO₂ retention is not usually aggravated by oxygen therapy in asthma)
- Salbutamol 5 mg or terbutaline 10 mg via an oxygen-driven nebuliser
- Ipratropium bromide 0.5 mg via an oxygen-driven nebuliser
- Prednisolone tablets 40-50 mg or IV hydrocortisone 100 mg or both if very ill
- No sedatives of any kind
- Chest radiograph only if pneumothorax or consolidation are suspected or patient requires IPPV

IF LIFE THREATENING FEATURES ARE PRESENT:

- Discuss with senior clinician and ICU team
- Add IV magnesium sulphate 1.2-2 g infusion over 20 minutes (unless already given)
- Give nebulised β_2 agonist more frequently e.g. salbutamol 5 mg up to every 15-30 minutes or 10 mg continuously hourly

SUBSEQUENT MANAGEMENT

IF PATIENT IS IMPROVING continue:

- 40-60% oxygen
- Prednisolone 40-50mg daily or IV hydrocortisone 100 mg 6 hourly
- Nebulised β_2 agonist and ipratropium 4-6 hourly

IF PATIENT NOT IMPROVING AFTER 15-30 MINUTES:

- Continue oxygen and steroids
- Give nebulised β_2 agonist more frequently e.g. salbutamol 5 mg up to every 15-30 minutes or 10 mg continuously hourly
- Continue ipratropium 0.5 mg 4-6 hourly until patient is improving

IF PATIENT IS STILL NOT IMPROVING:

- Discuss patient with senior clinician and ICU team
- IV magnesium sulphate 1.2-2 g over 20 minutes (unless already given)
- Senior clinician may consider use of IV β_2 agonist or IV aminophylline or progression to IPPV

MONITORING

- Repeat measurement of PEF 15-30 minutes after starting treatment
- Oximetry: maintain SpO₂ $> 92\%$
- Repeat blood gas measurements within 2 hours of starting treatment if:
 - initial PaO₂ < 8 kPa (60 mmHg) unless subsequent SpO₂ $> 92\%$
 - PaCO₂ normal or raised
 - patient deteriorates
- Chart PEF before and after giving β_2 agonists and at least 4 times daily throughout hospital stay

Transfer to ICU accompanied by a doctor prepared to intubate if:

- Deteriorating PEF, worsening or persisting hypoxia, or hypercapnea
- Exhaustion, feeble respirations, confusion or drowsiness
- Coma or respiratory arrest

DISCHARGE

When discharged from hospital, patients should have:

- Been on discharge medication for 24 hours and have had inhaled technique checked and recorded
- PEF $> 75\%$ of best or predicted and PEF diurnal variability $< 25\%$ unless discharge is agreed with respiratory physician
- Treatment with oral and inhaled steroids in addition to bronchodilators
- Own PEF meter and written asthma action plan
- GP follow up arranged within 2 working days
- Follow up appointment in respiratory clinic within 4 weeks

Patients with severe asthma (indicated by need for admission) and adverse behavioural or psychosocial features are at risk of further severe or fatal attacks

- Determine reason(s) for exacerbation and admission
- Send details of admission, discharge and potential best PEF to GP

Peak expiratory flow in normal adults

