

Guidelines for the management of asthma in adults in South Africa

Part II. Acute asthma

STATEMENT BY A WORKING GROUP OF THE SOUTH AFRICAN PULMONOLOGY SOCIETY

The morbidity and mortality caused by asthma can be attributed to three factors: underassessment of severity, failure on the part of both patients and their medical attendants to initiate treatment promptly, and undertreatment of exacerbations. Moreover, most exacerbations can be prevented by use of appropriate long-term treatment (*S Afr Med J* 1992; 81: 319-322). The present guidelines are for the care of acute asthma ('asthma attacks') and are intended to encourage a uniform approach to the management of exacerbations, whether of rapid or gradual onset, mild or severe. They have been developed on the basis of the best available evidence on the efficacy and safety of asthma drugs, and efforts have been made to ensure that recommendations are cost-effective and affordable, and may with little modification be applied in all locations: the home (as initial self-management), the clinic with modest facilities, doctors' surgeries, emergency departments and hospitals. The guidelines stress (i) assessment of severity; (ii) recognition of risk; and (iii) stepwise treatment based upon these assessments. Primary therapies are the repeated use of high doses of β_2 -agonists and early introduction of corticosteroids.

Specific goals of treatment are to: (i) relieve airway obstruction; (ii) relieve hypoxaemia; (iii) restore lung function to normal as rapidly as possible by reducing airway irritability; (iv) provide a suitable plan to avoid future relapse; and (v) provide a written plan of action to be followed early in future attacks.

Simplified management schemes for different locations are provided as addenda for ease of reference.

S Afr Med J 1994; 84: 332-338.

Definition and general comment

Acute asthma (the 'acute asthma attack') is usually an exacerbation of chronic asthma, and is characterised by worsening shortness of breath, cough, wheeze and chest tightness, or combinations of these symptoms, often with apparent non-responsiveness to usual bronchodilator treatment. It may range from mild to life-threatening and is always associated with a fall in expiratory airflow measured as peak expiratory flow (PEF) rate or forced expiratory volume in 1 second (FEV₁). The latter serve as more reliable indicators of severity of airflow obstruction than the severity of symptoms.

Patterns of onset

Exacerbations usually develop over hours or days, but occasionally occur precipitously over minutes, either spontaneously or after exposure to allergens, noxious agents or other trigger factors. Slow deterioration usually reflects failure of long-term management.

Aims of management

Most asthma attacks are preventable and prevention of even mild exacerbations is the goal of maintenance treatment. By contrast, the treatment of acute asthma is crisis- and hospital-orientated; besides carrying a greater risk of death, brain damage, barotrauma and other complications of resuscitation, it is also more costly. Fear, anxiety and disruption of normal activities are inevitable. Studies of asthma deaths have confirmed that fatal attacks are often associated with: (i) underestimation of the severity of the attack by patients, their relatives and their doctors, largely because of failure to make objective measurements; (ii) failure to initiate treatment promptly; and (iii) undertreatment of the exacerbation.

Aims of management of acute asthma are to prevent this morbidity and mortality by: (i) relieving airflow limitation as rapidly as possible; (ii) relieving hypoxaemia; (iii) restoring lung function to normal as soon as possible by treating airway inflammation and reactivity; (iv) providing a suitable plan to avoid future relapses; and (v) providing a written plan of action to be followed early in future attacks.

The current guidelines apply to acute asthma and not to chronic bronchitis and emphysema caused by cigarette smoking. Although some of the principles of treatment are similar, there are important differences. For example, in the latter, airflow obstruction is largely irreversible, and the free use of oxygen may precipitate carbon dioxide retention and worsen respiratory failure.

Recognition and assessment of severity of attacks

This assessment is based upon a variety of clinical signs and measurement of PEF or FEV₁ (in all cases) and, when available, assessment of blood gas status or oxygen saturation (Table I). The table serves only as a guideline for deciding the initial course of action.

Caution. Patients with severe or life-threatening attacks may not appear distressed and not all features of a severe attack may be present. The presence of any of these features indicates deterioration to the corresponding grade of severity. A severe grade should be assumed if the attack progresses quickly or if, on the basis of his/her history, the patient is considered to be at high risk of an asthma-related death.

PEF measurements

Lower PEF rates are most easily interpreted when expressed as a percentage of the predicted normal value for the individual (Table II) or of the patient's best value obtained previously when on optimal treatment. If neither of these is known, a decision must be made on the basis of the absolute value recorded, remembering that normal values vary with age, sex and height; older people, women and shorter people have a lower normal range. Values for PEF and FEV₁ expressed as percentages of the predicted normal are less useful in patients with chronically impaired lung function (Tables II and III).

Arterial blood gases

Arterial blood gas status should be measured in: (i) all patients with clinical features of severe or life-threatening asthma (including all in the high-risk category and with a PEF rate or FEV₁ < 50% of the predicted value or the patient's best); and (ii) oxygen saturation on pulse oximetry of less than 90%.

TABLE I.
Severity of asthma exacerbations

	Moderate	Severe/ life-threatening	Respiratory arrest imminent (if any of listed signs present)
PEF after initial bronchodilator*	Approx. 50 - 75%	< 50%	< 100 l/min
Breathlessness	Talking Prefers sitting	At rest Hunched forward	Exhaustion
Talks in	Phrases	Words	
Alertness	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate	Increased	Often > 30/min	
Accessory muscles and suprasternal retraction	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Loud	Usually loud	Absence of wheeze
Pulse/min	100 - 120	> 120	Bradycardia
Pulsus paradoxus (inspiratory fall in systolic blood pressure)	May be present 10 - 25 mmHg	Often present > 25 mmHg	Absence suggests respiratory muscle fatigue
PaO ₂ (on air)	> 8 kPa (60 mmHg)	< 8 kPa Possible cyanosis	Cyanosis
Paco ₂	< 6 kPa (45 mmHg)	> 6 kPa	
Sao ₂ (on air)	91 - 95%	< 90%	

Blood gases are advised for all patients admitted to hospital with features suggesting severe asthma.
*PEF expressed as percentage of normal value (read off Table II; according to age, sex and height), or the patient's best PEF value obtained when on optimal treatment.

TABLE II.
Predicted PEF (l/min)

Age (yrs)	25	30	35	40	45	50	55	60	65	70
Height (cm)										
Men										
160	534	521	508	495	482	469	457	444	431	418
165	552	539	527	514	501	488	475	462	449	436
170	571	558	545	532	519	506	493	480	468	455
175	589	576	563	551	538	525	512	499	486	473
180	608	595	582	569	556	543	530	517	504	492
185	626	613	600	587	574	562	549	536	523	510
190	644	632	619	606	593	580	567	554	541	528
Women										
145	367	358	349	340	331	322	313	304	295	286
150	383	374	365	356	347	338	329	320	311	302
155	400	391	382	373	364	355	346	337	328	319
160	416	407	398	389	380	371	362	353	344	335
165	433	424	415	406	397	388	379	370	361	352
170	449	440	431	422	413	404	395	386	377	368
180	482	473	464	455	446	437	428	419	410	401

Quanjer PH, ed. Working Party of European Community for Coal and Steel. Summary of recommendations: standardisation of lung function tests. *Bull Eur Physiopath Resp* 1983; 19: suppl 5, 7-10.

TABLE III.
Predicted FEV₁ (l)

Men	FEV ₁ = 4,301 x H - 0,029 x A - 2,492
Women	FEV ₁ = 3,953 x H - 0,025 x A - 2,604

H is height in metres; A is age in years.

Quanjer PH, ed. Working Party of European Community for Coal and Steel. Summary of recommendations: standardisation of lung function tests. *Bull Eur Physiopath Resp* 1983; 19: suppl 5, 7-10.

The determination of arterial blood gas status must not delay initiation of treatment.

Assessment of high risk

Patients with the following history are at high risk of respiratory arrest and any attack must be considered severe: (i) current use of, or recent withdrawal from, systemic corticosteroids; (ii) emergency care and/or hospitalisation for asthma in the past year or recent treatment for acute asthma; (iii) previous resuscitation and intubation for acute severe asthma; and (iv) previous sudden severe attack/s with few or no warning features in spite of regular treatment. When these attacks occur repeatedly, the condition is termed 'brittle asthma'.

Management of exacerbations

Home management of exacerbations

(Fig. 1)

All patients with chronic asthma should have a written plan of action outlining how to recognise warning signs of deteriorating asthma, adjust treatment and obtain emergency medical care.

Warning signs of worsening asthma

These are: (i) increased need for and use of a bronchodilator; (ii) apparently decreased effectiveness of a bronchodilator (partial or short-lived response); (iii) symptoms of asthma at night (disturbed sleep); (iv) early morning waking ('morning dipping'); (v) persistent cough; (vi) impaired ability to perform daily activities; (vii) increased diurnal variation in PEF rate; and (viii) a downward trend in PEF readings.

Adjusting treatment

The scheme of advice given to patients is presented in diagrammatic form in Fig. 1. Initial treatment involves immediate use of repeated high doses of inhaled β_2 -agonists. Where the response is incomplete or short-lived, additional therapy is indicated. However, even when the response is good, full recovery is often gradual and modification of maintenance asthma medication is necessary.

Emergency medical care

Patients should seek medical help without delay if: (i) they have been identified as at high risk — they should be aware of this; (ii) the exacerbation is severe (Table I); (iii) the response to a bronchodilator is not prompt or is not sustained for at least 3 hours; (iv) there is no sustained improvement in PEF rate within 4 - 6 hours of corticosteroid treatment; or (v) there is further deterioration.

Management of exacerbations by medical/nursing personnel

The observations that follow apply to treatment given by medical and/or nursing personnel at clinics, consulting rooms, emergency departments or hospitals.

Principles of treatment

1. Begin treatment immediately.

2. **Oxygen.** Use the highest concentration mask and set at a high flow rate. Oxygen nasal cannulas may be used if a mask is not tolerated but they only deliver low concentrations of oxygen. Retention of carbon dioxide is not aggravated by treatment with oxygen in patients with acute severe asthma. The target oxygen saturation is 90% or more.

3. Primary drug treatment for acute asthma.

This entails: (i) repeated use of high doses of inhaled β_2 -agonists to relieve bronchospasm; and (ii) corticosteroids (usually by mouth or intravenously) to reduce bronchial inflammation and hyperreactivity.

High doses of inhaled β_2 -agonists are given by nebulisation or through a large spacer device attached to a metered dose inhaler (MDI). To ensure oxygenation, oxygen-driven nebulisation is recommended over air-driven nebulisation. The initial treatment is one dose

every 20 minutes for 1 hour. Dosing of nebuliser solutions (diluted 1:4 with normal saline) is as follows: fenoterol 1 mg, hexoprenaline 0,25 mg or salbutamol 5 mg. Subsequently, hourly administration or even continuous nebulisation should be given if the episode is severe.

If a nebuliser is not available, a large dose of β_2 -agonist can be given via multiple actuations of a MDI into a large spacer device up to the total dose delivered by nebuliser. The spacer devices should have a volume greater than 500 ml. Commercial models include Volumatic (Allen and Hanbury) and Nebuhaler (Astra), and doses may be administered through a mouthpiece or specially designed mask. Where these spacers are not available, a plastic bottle of similar size, with the base cut out to fit snugly over the mouth and nose, may be used. The patient is instructed to take several deep breaths from the spacer after each pair of actuations. In the home 4 - 10 puffs are recommended but may be repeated within minutes if the response remains poor, and while transport to a doctor or clinic is being arranged (Fig. 1). In the emergency room a total of 20 or more puffs (with a limit of 50 puffs) may be given.

The early use of *systemic corticosteroids* is strongly recommended; their underuse has been linked to asthma deaths and early relapse. They speed up resolution and prevent relapse and should be commenced without delay, as they take at least 4 hours to produce clinical improvement. An advantage of intravenous over oral treatment has not been demonstrated and the former is considerably more expensive. However, because gastro-intestinal absorption is suspect or doubtful in severe attacks, the intravenous route is advised, at least for the initial dose to ensure early peak dosing.

Corticosteroids benefit all patients but are essential when: (i) the exacerbation is moderate or severe; (ii) the initial inhaled β_2 -agonist dose fails to achieve significant improvement; (iii) the patient develops acute asthma despite the regular use of oral corticosteroids; or (iv) previous exacerbations required oral corticosteroids.

Usual doses are prednisolone, methylprednisolone or prednisone 30 - 60 mg as a single oral daily dose (usually in the morning) or hydrocortisone 200 mg intravenously 4 - 6-hourly, or methylprednisolone 125 mg twice daily intravenously. Corticosteroids should be continued for a week after all symptoms of

HOME ACTION PLAN FOR ATTACKS OF ASTHMA

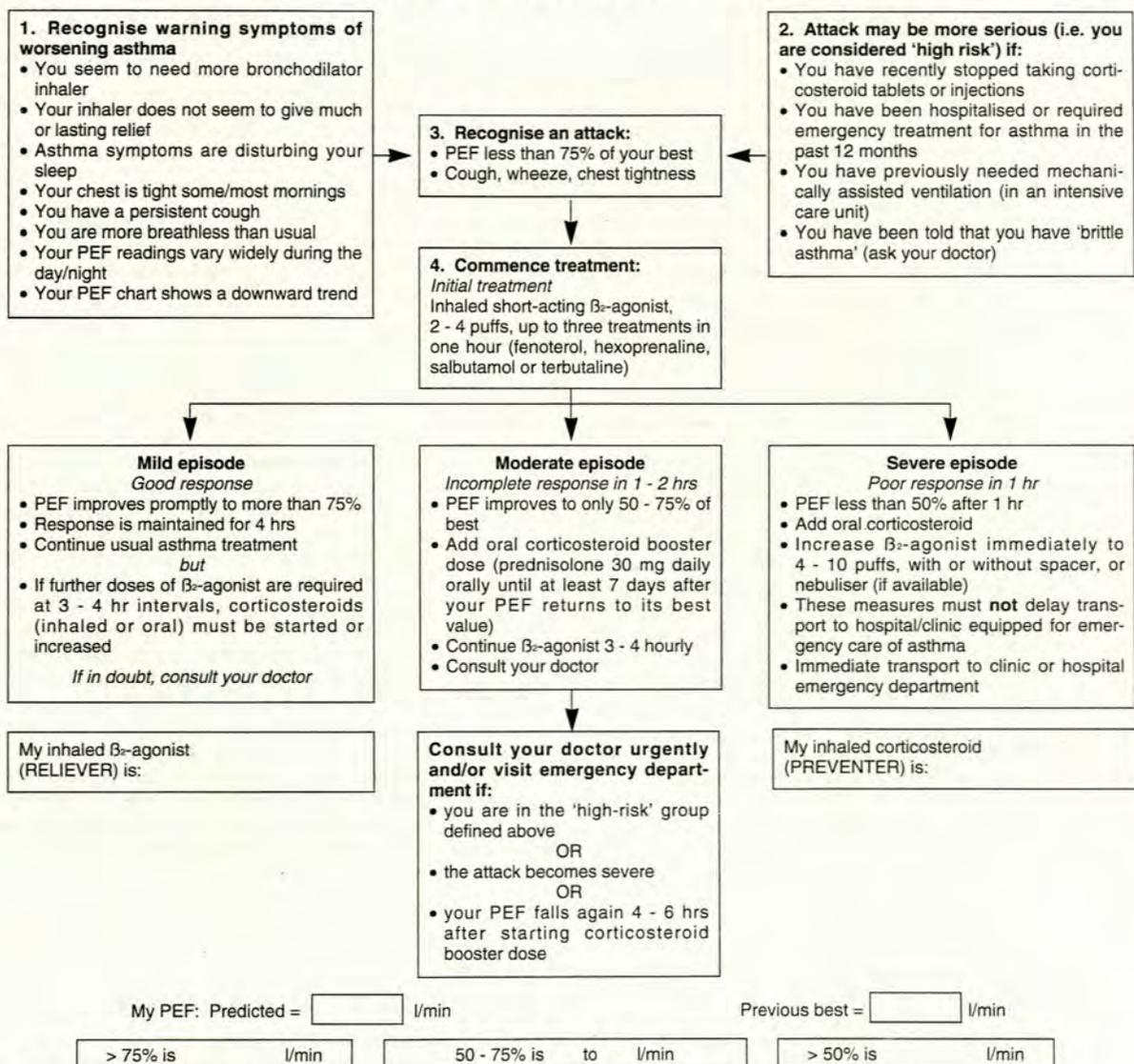


FIG. 1. Scheme for self-management of exacerbations of asthma at home. This scheme will be made available in a card format for distribution to patients, and is a guide to be followed during an acute asthma attack.

asthma have disappeared, i.e. at least 10 days. By that time the long-term preventive treatment plan should have been commenced or re-established. In patients on inhaled steroid therapy, short courses of oral corticosteroids may be stopped abruptly without 'tapering' of the dose, as they are not associated with sustained adrenal suppression. However, when a patient has been taking oral steroids constantly or frequently, slow reduction of the dose is advisable. Due attention should also be paid to pre-existing conditions that might be adversely affected by higher doses, e.g. peptic ulceration and diabetes mellitus.

4. Additional bronchodilators. *Anticholinergic drugs* have a different mechanism of action from that of β_2 -agonists and provide additional bronchodilatation in acute asthma. They should not be used alone, but may be used in combination with or alternate with the latter, either by nebulisation or in high doses from a MDI attached to a spacer. Their use is not associated with significant side-effects and is preferred to other bronchodilators. They should be used in all patients with severe episodes that require hospitalisation and who fail to improve on the standard regimen, as well as in those intolerant of high doses of β_2 -agonists. The usual dose is 0,5 mg ipratropium bromide nebuliser solution 4-hourly or 2 puffs from a MDI repeated to a total of 20 puffs every 4 hours. It may be discontinued when it is clear that the patient is responding.

Intravenous theophylline is a significantly less effective bronchodilator than β_2 -agonists and ipratropium bromide. Serious limitations to its use include: (i) a high incidence of acute nausea, vomiting and other side-effects when administered rapidly; (ii) serious side-effects if a patient has previously been on long-acting slow-release theophyllines and has therefore been rendered acutely toxic; (iii) the fact that additional benefit beyond that obtained with nebulised β_2 -agonist and steroid alone has not been clearly demonstrated; and (iv) the fact that standard recommended doses for intravenous infusions of theophylline are unreliable given the large number of factors that influence its metabolism. Monitoring of blood levels is desirable if not essential. Lower doses are required in patients with liver disease or heart failure and in those taking cimetidine, ciprofloxacin, erythromycin and most other macrolide antibiotics. Higher doses are required in smokers. Although the unit cost of aminophylline is lower than for nebulised β_2 -agonists and ipratropium bromide, this is offset by the additional cost of needles, syringes, infusion sets, blood level monitoring and treatment of complications.

The dose regimen for aminophylline is as follows: (i) loading dose 6 mg/kg over 30 minutes (this must be withheld, or the dose must be halved in patients on oral theophyllines); (ii) average maintenance dose: 0,6 mg/kg/h = approximately 1 000 mg/24 h (see (v) and (vi) for dosage adjustments); (iii) never use more than 1 000 mg/d without monitoring blood levels; (iv) individual clearance in normal adults varies widely, i.e. individual dosages vary from 300 to 3 000 mg/d; (v) increase dose by one-third in smokers and patients on phenytoin therapy; and (vi) decrease dose by one-third in patients with congestive cardiac failure, the elderly, patients with liver disease and patients on most macrolide antibiotics, ciprofloxacin or cimetidine.

Beta-2-agonists administered *intramuscularly*, *subcutaneously* or by *continuous intravenous infusion* have not been shown to be superior to inhaled β_2 -agonists and are associated with more side-effects. They may be considered in severe attacks where nebulised β_2 -agonists fail to achieve a sustained response. A loading dose, followed by constant infusion is recommended, the rate of which is adjusted according to improvement in the PEF rate. Side-effects include tachycardia and hypokalaemia. Serum potassium levels should be measured at least

daily. The infusion should be stopped when the patient shows sustained improvement (and should usually not be continued longer than 24 hours).

Usual doses are: salbutamol 0,5 mg intramuscularly or subcutaneously, or 0,25 mg slowly intravenously. This may be repeated 4-hourly as required or once followed by an infusion of 3 - 20 μ g/min of a solution 5 mg/5 ml diluted in 500 ml dextrose water or normal saline.

Adrenaline injected subcutaneously is indicated for acute treatment of anaphylaxis, angio-oedema and, occasionally, for asthma when severe exacerbations are not relieved by inhaled β_2 -agonists and ipratropium bromide. Being less β_2 -agonist-specific, it has several side-effects, and its advantage over β_2 -specific agonists is doubtful.

The following do not form part of the routine management of acute exacerbations:

1. Antibiotics are indicated only for patients with fever and purulent sputum (caused by polymorphs, not eosinophils) that suggest bacterial infection.

2. Inhaled mucolytics should not be used as they worsen cough and airflow obstruction.

3. Sedation is contraindicated because of its respiratory depressant effect.

4. Antihistamines have no role in the treatment of exacerbations.

5. Percussive chest physiotherapy is not beneficial and may actually provoke bronchospasm and worsen the attack.

6. Hydration with large volumes of fluids is unnecessary in adults and older children.

Periodic assessments

A nurse or doctor should remain with the patient for at least 15 minutes after admission and until improvement is observed. The following should be monitored at frequent intervals. **PEF** should be measured 15 - 30 minutes after starting treatment, at 2 hours and thereafter, before and after each treatment with nebulised β_2 -agonist (at least 4 times daily) throughout the admission.

Other clinical features for assessing severity of asthma (Table I) should be measured hourly in severe cases, 4-hourly in those showing a satisfactory response.

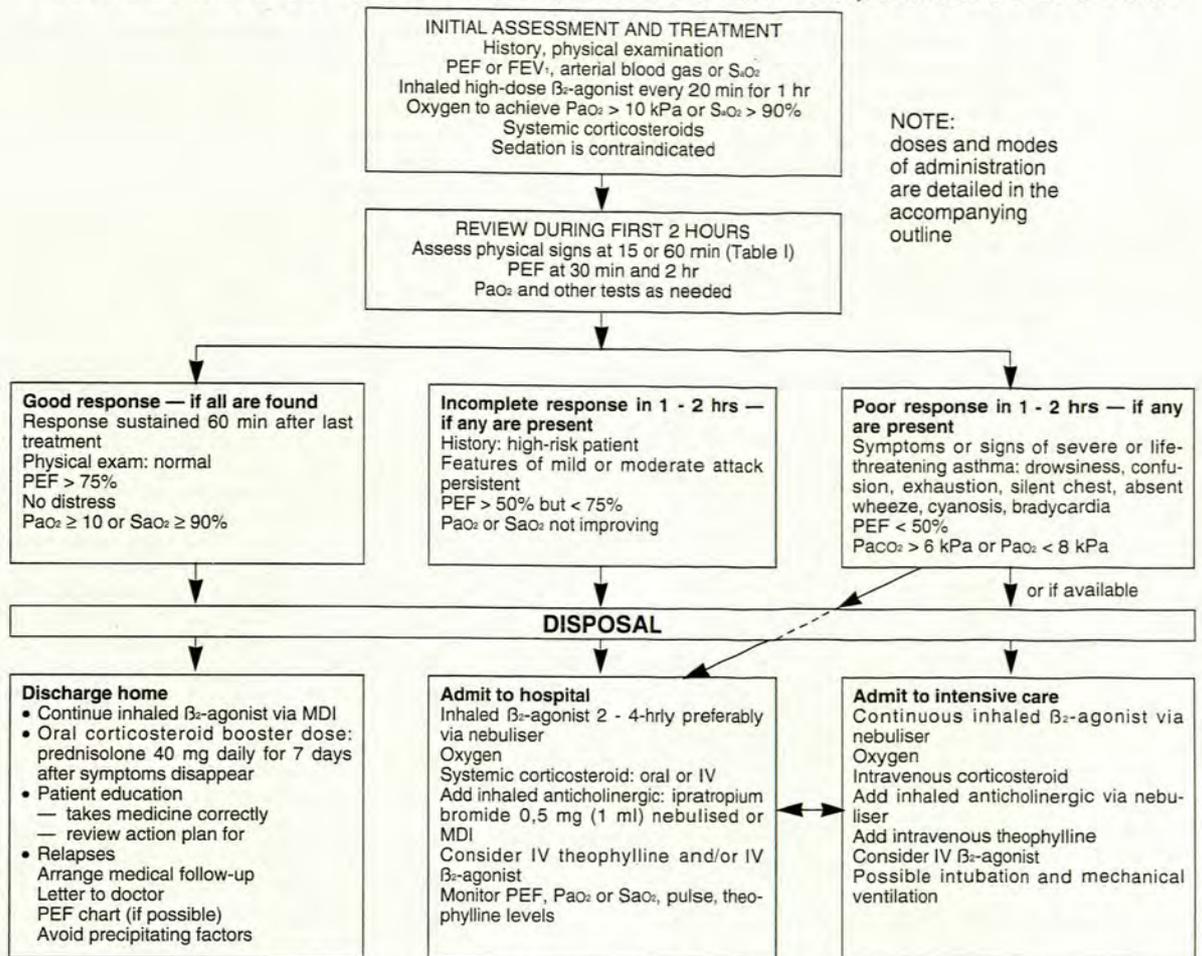
Blood gas measurements should be repeated only if the patient's condition appears to be deteriorating, or if it fails to improve after 2 or 4 hours in patients in whom the initial P_{aO_2} was low and initial P_{aCO_2} was normal or raised. **Anxiety** should be relieved by verbal reassurance and care. Where treatments have similar efficacy and safety, non-invasive and non-painful methods should be used. Thus, inhaled or oral β_2 -agonists and corticosteroids are preferred over intravenous or subcutaneous therapy and pulse oximetry is preferred over arterial blood gas measurements. **Routine chest radiography** is not necessary, but should be performed if pneumothorax, atelectasis, pneumonia or pulmonary oedema is suspected, or if the patient fails to respond promptly to treatment. **Plasma electrolyte** and **urea** values should be measured during prolonged attacks and when the development of hypokalaemia is possible (e.g. during systemic β_2 -agonist use).

The need for other investigations e.g. blood counts, electrocardiography, should be judged on an individual basis.

Criteria for admission to hospital (longer stay)

Both medical and social factors must be considered. When in doubt, admit. Factors suggesting the need for hospitalisation include: (i) inadequate response to therapy within 1 - 2 hours of treatment (Fig. 2); (ii) per-

MANAGEMENT OF EXACERBATION OF ASTHMA IN PRACTICE ROOMS, CLINICS AND HOSPITALS



SYNOPSIS OF DRUGS AND DOSES IN ACUTE ASTHMA

OXYGEN
Use highest concentration mask (60% or higher)

BETA-2-AGONISTS — INHALED
Via NEBULISER (preferably oxygen-driven)
dose: fenoterol 1 mg 1 ml in 4 ml sterile normal saline
hexoprenaline 0,25 mg
salbutamol 5 mg
frequency: initially every 20 minutes for 1 hour
2 - 4-hourly while in hospital/surgery
Via MDI and SPACER
dose: fenoterol 100 µg/puff } several deep breaths
hexoprenaline 100 µg/puff } from spacer after
salbutamol 100 µg/puff } each 2 puffs to total
terbutaline 250 µg/puff } of 20 to 50 puffs
frequency: initially every 20 minutes for 1 hour
2 - 4-hourly while in hospital
spacers: minimum volume 500 ml
Volumatic (Allen & Hanbury)
Nebuhaler (Astra)
plastic bottle with base removed to fit snugly onto face

CORTICOSTEROIDS
Initial dose: prednisone, methylprednisolone or prednisolone 30 mg oral
or hydrocortisone 200 mg IV
or methylprednisolone 125 mg IV
further doses:
Initial good response: continue daily oral dose for 7 days after complete recovery. No tapering necessary unless on long-term oral dose
Partial response: continue daily oral dose or hydrocortisone 200 mg IV 4 - 6-hourly or methylprednisolone 125 mg IV twice daily
Poor response: continue IV doses

IPRATROPIUM BROMIDE
Via NEBULISER (preferably oxygen-driven)
dose: 0,5 mg (2 ml in 3 ml sterile normal saline)
frequency: 4-hourly
Via MDI and SPACER
dose: 20 µg/puff; up to 20 puffs
frequency: 4-hourly

AMINOPHYLLINE — INTRAVENOUS
loading dose: 6 mg/kg over 30 minutes (10 ml ampoule = 250 mg)
Withhold or give only half to patients on oral theophyllines
maintenance dose: 0,6 mg/kg/hour (± 1 000 mg/24 hours)
Increase by one-third (0,9 mg/kg/hour) in smokers and patients taking phenytoin
Decrease by one-third in congestive cardiac failure, elderly, liver disease, treatment with macrolide antibiotics, ciprofloxacin or cimetidine.
monitor: blood levels daily and adjust dose

BETA-2-AGONISTS — INTRAVENOUS
Initial dose: salbutamol 0,25 mg IV slowly
maintenance dose: salbutamol 3 - 20 µg/minute (5 mg diluted in 500 ml dextrose water or normal saline)
duration: until sustained improvement (usually less than 24 hours)
monitor: serum K⁺ daily

FIGS 2 AND 3.

Extracts from a wall poster to be distributed to clinics, hospitals and doctors' surgeries, that contains a summary of a scheme recommended for the management of acute asthma attacks in these settings.

sistent severe airflow limitation (PEF rate less than 50% of predicted or personal best); (iii) past history of severe asthma, particularly if hospitalisation was required; (iv) recurrence after recent exacerbation; (v) presence of high-risk factors; (vi) prolonged symptoms before current emergency room visit; or (vii) any of the following social and personal factors: inadequate access to medical care and medication, difficult home conditions, difficulty obtaining transport to hospital in the event of further deterioration, difficulty following asthma management plan.

An overnight stay is advised when the patients are seen in the afternoon or evening rather than earlier in the day.

Criteria for admission to an intensive care unit

Patients are considered in need of intensive care when either of the following is present: (i) little response to initial therapy at clinic or hospital (Fig. 2); or (ii) signs of imminent respiratory arrest, such as cyanosis, exhaustion, drowsiness, confusion, silent chest, bradycardia, rising PaCO₂ and persistent acidosis (Table I). It is preferable to err on the side of caution.

Not all patients admitted to the intensive care unit need intubation and ventilation. Although it is desirable to avoid ventilation, delaying tracheal intubation until the patient suffers respiratory and/or cardiac arrest is a serious error of judgement. Intubation is indicated where there is progressive deterioration in clinical features despite optimal therapy, the patient is exhausted and/or acidotic and/or the PaCO₂ continues to rise. As intubation of the distressed asthmatic requires considerable skill, it should be unhurried and performed by the most experienced person available. Details of the ventilatory management of asthmatics will not be considered further in this report.

Management prior to discharge

Early discharge after moderate episodes of acute asthma is indicated where, as defined in Fig. 2, exacerbations respond rapidly to initial treatment, provided that the patient's PEF or FEV₁ has returned to 75% or more of predicted or personal best, physical examination is normal and the patient feels both undistressed and recovered. Patients admitted for severe attacks should not be discharged until their symptoms have cleared, lung function tests stabilised or returned to their usual best (PEF > 75%, diurnal variability < 25%) and nocturnal symptoms have been alleviated. Diurnal variability is calculated as follows: highest PEF minus lowest

PEF in each 24-hour period divided by highest PEF and multiplied by 100.

All patients considered ready for discharge require the following: (i) regular inhaled β_2 -agonists (4-hourly). Change from nebuliser to standard MDIs 24 - 48 hours before discharge. Check patients' inhaler technique; (ii) to commence or continue taking inhaled anti-inflammatory drug, usually a corticosteroid; (iii) to continue the booster course of oral corticosteroid for at least a week after symptoms of asthma have disappeared, or until the follow-up visit; (iv) an appointment with their family practitioner or usual physician within days or, at most, 2 weeks, and a discharge note stating details of treatment and discharge PEF rate; (v) to have ready access to emergency medical care should a recurrence occur within days; (vi) a self-management plan with instructions about recognising deterioration, self-adjustment to treatment, early notification of their doctor and self-referral for emergency treatment. Some patients may benefit from provision of a peak flow meter to guide their self-management plan; and (vii) a review of the circumstances leading to this admission, and sound advice in this regard. Was there an avoidable precipitating cause, e.g. upper respiratory tract infection or an inhaled agent in the home or workplace? Give advice on trigger factors. What was the pattern of deterioration: acute and sudden or with recognisable slow deterioration? Did the patient (and/or relatives) react appropriately? Was the patient compliant with regular treatment? Was medical management in the emergency room and hospital appropriate?

Review the asthma action plan to ensure that doses of regular medications are appropriate.

These guidelines are modelled on *The International Consensus Report on Diagnosis and Treatment of Asthma* published by the US Department of Health and Human Services (publication No. 92-3091, June 1992) and *Acute Severe Asthma in Adults and Children* by the British Thoracic Society (*Thorax* 1993; 48: suppl, S12-S24). The South African Pulmonology Society gratefully acknowledges the authors and publishers of these reports.

Rapporteur: E. D. Bateman

Planning Group: M. Plit (Convenor), E. D. Bateman, J. R. Joubert

Other members of Consensus Seminar: M. R. Becklake, K. R. Chapman, C. Feldman, M. Greenblatt, G. Irsigler, M. C. Kamdar, U. G. Lalloo, A.-M. le Roux, F. le Roux, F. Muller, J. O'Brien, D. Pansegrouw, A. Promnitz, R. I. Raine, G. Ras, G. Richards, B. W. van de Wal, S. Zwi