

INTRODUCTION

Asthma is one of the commonest of all medical conditions. It is caused by a chronic inflammation of the bronchi, making them narrower. The muscles around the bronchi become irritated and contract, causing sudden worsening of the symptoms. The inflammation can also cause the mucus glands to produce excessive sputum which further blocks the air passages. These guidelines are concerned with the acute asthma attack.

HISTORY

The patient may well have a history of increased wheezy breathlessness, often worse at night or in the early morning, associated either with infection, allergy or exertion as a trigger. They are usually a known asthmatic and may well be on regular inhaler therapy for this. They may well have used their own treatment inhalers and in some cases will have used a home based nebuliser.

If a patient is suffering a first episode of 'asthma' always consider an inhaled foreign body as a differential diagnosis.

ASSESSMENT¹

Assess **ABCD's**:

Asthma usually presents to the ambulance service in one of two forms (*see Table 1*).

Table 1 – Two forms of asthma presentation

Life Threatening	Acute Severe
- exhaustion - confusion - coma - silent chest - cyanosis - feeble respiratory effort - bradycardia - hypotension - peak flow <33% of predicted best value - SpO₂ <92%	- unable to complete sentences in one breath - respiratory rate >25 (adult) - pulse >110 beats per minute - peak flow 33%-50% of predicted best value

Assess for any LIFE-THREATENING features

If **any** of these features are present, **start correcting A and B problems then transfer to nearest suitable receiving hospital** commencing oxygen (O₂) immediately at the patient side.

Provide a hospital alert message / information call.

Those with life threatening asthma may need paralysing and ventilating if they fail to respond to treatment. Rapid transfer to hospital on **blue lights** is therefore extremely important.

En-route continue patient **MANAGEMENT**, (*see below*) providing any other necessary interventions, including nebulisation, steroids etc.

If no **TIME CRITICAL** features are present:

- assess for features of acute severe asthma
- consider the benefits of treatment en-route to hospital unless the patient has a history of full recovery and subsequent refusal of transfer to further care
- any patient who is transferred to hospital requires at least O₂ and nebuliser treatment en-route
- remember that the risk of death in the group of asthmatics previously admitted to hospital with an acute attack is significant.

MANAGEMENT OF ASTHMA²⁻⁴

Follow medical emergencies guideline, remembering to:

Start correcting:

- **AIRWAY**
- **BREATHING**
- **CIRCULATION**
- **DISABILITY** (mini neurological examination)
- administer high concentration oxygen (O₂)⁵ (**refer to oxygen guideline for administration and information**) via a non-re-breathing mask, using the stoma in laryngectomy and other neck breathing patients, to ensure an oxygen saturation (SpO₂) of >95%, except in patients with chronic obstructive pulmonary disease (COPD) (**refer to COPD guideline**)
- commence transfer to further care.

Specifically:

check peak flow if practicable – note the best of three readings.

- administer **salbutamol**⁶⁻⁹ via O₂ driven nebuliser, running at 6-8 litres per minutes (**refer to salbutamol drug protocol for dosages and information**). In acute severe or life-threatening cases **ipratropium bromide** (**refer to ipratropium bromide drug protocol for dosages and information**) should be added to the salbutamol. Continue high concentration O₂ after nebulisation
- in cases of hypoventilation in-line nebulisation with a bag-valve-mask (BVM) device, where appropriate using the stoma in laryngectomy and other neck breathing patients and suitable nebuliser attachment should be considered
- monitor using ECG and pulse oximeter
- obtain IV access if possible (**DO NOT** delay transfer to further care)
- if no clinical improvement after 5-10 minutes, administer further salbutamol via nebuliser and consider continuous nebulised salbutamol. Ipratropium bromide should be administered if not given earlier
- repeat or continuous nebulised salbutamol may be given until arrival at hospital or side effects become clinically significant (extreme tachycardia >140 beat per minute in adults, tremors etc.)
- assess chest to exclude evidence of pneumothorax

NOTE: remember the very rare complication in severe asthma of bilateral pneumothoraces

- re-assess to evaluate any improvement in peak flow or improvement in air entry on chest assessment
- administer **hydrocortisone** (**refer to hydrocortisone drug protocol for dosages and information**) IV where there is a delay getting to hospital of 30 minutes or more. Although steroids take some time to take effect, the sooner they are administered the better.

LIFE-THREATENING ASTHMA

A small minority of cases may not respond to O₂ and nebuliser therapy. In these cases the use of subcutaneous or intramuscular epinephrine should be considered where:

- the patient is suffering from life threatening asthma
- ventilation is failing
- deterioration continues despite O₂ and continuous nebulised salbutamol.

This treatment should be reserved for the most serious cases and is NOT intended to be used as a matter of routine due to its arrhythmogenic properties.

Drug Therapy:

- administer **adrenaline**⁶⁻⁸ (**refer to epinephrine drug protocol for dosage and administration**)
- consider salbutamol (**refer to salbutamol drug protocol for dosage and administration**)
- consider ipratropium bromide (**refer to ipratropium bromide drug protocol for dosage and administration**).

Asthmatic patients do not have hypoxic drive and need high concentration O₂ therapy and nebulisation as described earlier.

ADDITIONAL INFORMATION

The obstruction and subsequent wheezing are caused by three factors within the bronchial tree:

1. increased production of bronchial mucus
2. swelling of the bronchial tube mucosal lining cells
3. spasm and constriction of bronchial muscles.

These three factors conspire to cause blockage and narrowing of the small airways in the lung. Because inspiration is an active process involving the muscles of respiration, the obstruction of the airways is overcome on breathing in. Expiration occurs with muscle relaxation, and is severely delayed by the narrowing of the airways in asthma. This generates the wheezing on expiration that is characteristic of this condition.

Medical Emergencies

The obstruction in its most severe form can be **TIME CRITICAL** and some **2,000 people a year die as a result of asthma**. In adults, asthma may often be complicated and mixed in with a degree of bronchitis, especially in smokers. This can make the condition much more difficult to treat, both routinely and in emergencies. The majority of asthmatic patients take regular "preventer" and "reliever" inhalers.

Asthma is managed with a variety of inhaled and tablet medications. Inhalers are divided into two broad categories (preventer and reliever). The preventer inhalers are normally anti-inflammatory drugs and these include steroids and other milder anti-inflammatory such as Tilade. The common steroid inhalers are beclomethasone (Becotide), budesonide (Pulmicort) and fluticasone (Flixotide).

These drugs act over a period of time on the lung to reduce the inflammatory reaction that causes the asthma. Regular use of these inhalers often eradicates all symptoms of asthma and allows for a normal lifestyle.

Treatment (reliever) inhalers include salbutamol (Ventolin), terbutaline (Bricanyl) and ipratropium bromide (Atrovent). These inhalers work rapidly on the lung to relax the smooth muscle spasm when the patient feels wheezy or tight chested. They are used in conjunction with preventer inhalers. Inhalers are often used now through large plastic spacer devices, such as the Volumatic. This allows the drug to spread into a larger volume and allows the patient to inhale it more effectively.

In mild and moderate asthma attacks some patients may be treated with high doses of “relievers” through a spacer device. This has been shown to be as effective as giving a salbutamol nebuliser.

Peak Flow Metering

Peak flow is a rapid measurement of the degree of obstruction in the patient's lungs. It measures the maximum flow on breathing out, or expiring and therefore can reflect the amount of airway obstruction. Many patients now have their own meter at home and know what their normal peak flow is. Clearly, when control is good, their peak flow will be equivalent to a normal patient's measurement, but during an attack it may drop markedly.

Patients using a peak flow meter for the first time since 2004 will be given a new EU-scale meter. Existing asthmatic patients who require a replacement meter may notice that their readings have changed.

Peak Expiratory Flow (PEF) readings obtained on an EU-scale meter will be more accurate than those from a Wright scale meter, because changes in airflow will result in PEF readings changing uniformly for the whole range of the meter. The Wright scale has been previously noted to over-represent changes in airflow in the mid-range, and under represent changes in the low and high ranges.

Correcting these small inaccuracies results in PEF readings that are different – until the new EN 13826 standard meters are used for all PEF measurements, it will be important to note which scale has been used with the patient.

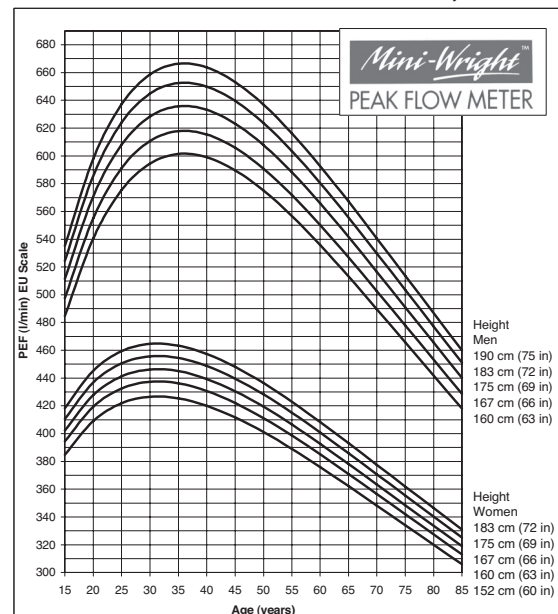
The new peak flow meters have a similar appearance to the old meters, but the scale (the part of the meter that you read the PEF value from) will have changed. If you use a Mini-Wright, the EU-scale will be a different

colour – blue text printed on a yellow background. Apart from the scale, the new Mini-Wright behaves and handles as reliably as the old meter.

It is important to recognise that if a patient knows their normal peak flow using one scale, that this may not be comparable to readings taken using a meter that has a different scale.

PEAK EXPIRATORY FLOW RATE - NORMAL VALUES

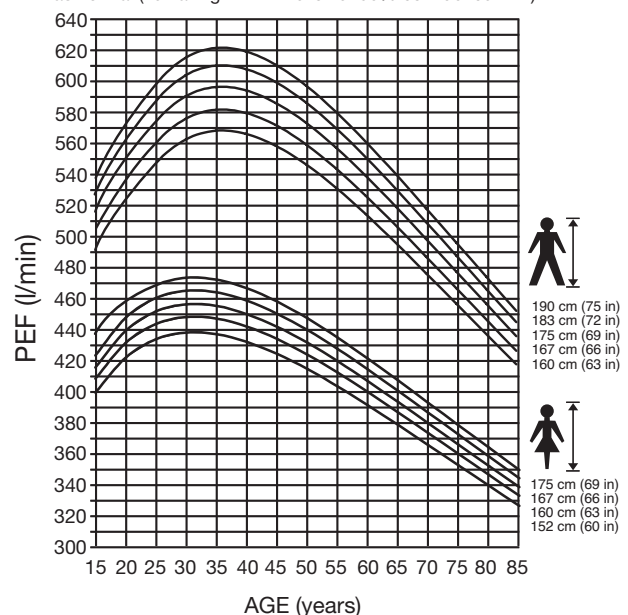
For use with EU/EN13826 scale PEF meters only



Adapted by Clement Clarke for use with EN13826 / EU scale peak flow meters from Nunn AJ Gregg I, Br Med J 1989;298;1068-70

For use with Wright " scale peak flow meters

A value of up to 80 litres/min below the mean can be regarded as normal (i.e. falling within the lower 95% confidence limit)



From Nunn AJ Gregg I, Br Med J 1989;298;1068-70

Figure 1 – peak flow charts

Steroid therapy.¹⁰⁻¹⁷

Steroids need to be given early in an acute asthma attack and can be given intravenously as hydrocortisone.

Key Points – Asthma

- Asthma is a common life threatening condition.
- Its severity is often not recognised.
- Accurate documentation is essential.
- Peak flow can be measured on more than one scale.
- A silent chest is a pre-terminal sign.

REFERENCES

- ¹ Rebuck AS, Read J. Assessment and management of severe asthma. *Am J Med* 1971; 51: 788-98. *American Journal of Medicine* 1971;51:788-98.
- ² British Thoracic Society, Scottish Intercollegiate Guidelines Network. The BTS/SIGN British Guideline on the Management of Asthma. *Thorax* 2003;58(Supplement 1):i1-i94.
- ³ British Thoracic Society, National Asthma Campaign, Royal College of Physicians of London in association with the General Practitioner in Asthma Group, The British Association of Accident and Emergency Medicine, The British Paediatric Respiratory Society, Health RCoPaC. The British guidelines on asthma management 1995 review and position statement. *Thorax* 1997;52(Suppl 1):S1-S21.
- ⁴ British Thoracic Society. Guidelines on the management of asthma: statement by the British Thoracic Society, British Paediatric Association, the Research Unit of the Royal College of Physicians of London, the Kings Fund Centre, the National Asthma Campaign, the Royal College of General Practitioners, the General Practitioners in Asthma Group, the British Association of Accident and Emergency Medicine, and the British Paediatric Respiratory Group following a meeting at the Royal College of Physicians of London on 4 and 5 June 1992. *Thorax* 1993;48(Supplement):S1-S24.
- ⁵ Cochrane GM. Acute severe asthma: oxygen and high dose beta agonist during transfer for all? *Thorax* 1995;50:1-2.
- ⁶ Salmeron S, Brochard L, Mal H. Nebulized versus intravenous albuterol in hypercapnic acute asthma: a multicentre, double-blind, randomized study. *American Journal of Respiratory Critical Care Medicine* 1994;149:1466-70.
- ⁷ Lawford P, Jones BM, Milledge JS. Comparison of intravenous and nebulised salbutamol in initial treatment of severe asthma. *BMJ* 1978(1):84.
- ⁸ Crompton GH. Nebulized or intravenous beta 2 adrenoceptor agonist therapy in acute asthma? *European Respiratory Journal* 1990(3):125-6.
- ⁹ Fergusson RJ, Stewart CM, Wathen CG. Effectiveness of nebulised salbutamol administered in ambulances to patients with severe acute asthma. *Thorax* 1995;50:81-2.
- ¹⁰ Rowe BH, Spooner C, Ducharme FM. Early emergency department treatment of acute asthma with systemic corticosteroids (Cochrane review). *The Cochrane Library* 2001;3.
- ¹¹ Rowe BH, Spooner C, Ducharme FM. Corticosteroids for preventing relapse following acute exacerbations of asthma (Cochrane review). *The Cochrane Library* 2001;3.
- ¹² Fanta CH, Rossing TH, McFadden ER. Glucocorticoids in acute asthma. A critical controlled trial. *Am J Med* 1983; 74: 845-51. *American Journal of Medicine* 1983;74:845-51.
- ¹³ Fiel SB, Swartz MA, Glanz K, Francis ME. Efficacy of short term corticosteroid therapy in outpatient treatment of acute bronchial asthma. *American Journal of Medicine* 1983;75:259-62.
- ¹⁴ Chapman KR, Verbeek PR, White JG, Rebuck AS. Effect of a short course of prednisone in the prevention of early relapse after the emergency room treatment of acute asthma. *N Engl J Med* 1991;324:788-94.
- ¹⁵ Ratto D, Alfaro C, Sipsey J. Are intravenous corticosteroids required in status asthmaticus? *JAMA* 1988;260:527-29.
- ¹⁶ Stein LM, Cole RP. Early administration of corticosteroids in emergency room treatment of acute asthma. *Annals of Internal Medicine* 1990;112:822-27.
- ¹⁷ MRC Sub-Committee on Clinical Trials. Controlled trial of the effects of cortisone acetate in status asthmaticus. Report to the Medical Research Council by the sub committee on clinical trials. *Lancet* 1956;271(6947):803-6.

METHODOLOGY

Refer to methodology section.