ASTHMA

Advanced Life Support

ERC GUIDELINES 2015 EDITION

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Worldwide, approximately 300 million people of all ages and ethnic backgrounds have asthma with a high prevalence in some European countries (United Kingdom, Ireland and Scandinavia). Annual worldwide deaths from asthma have been estimated at 250,000. The death rate does not appear to be correlated with asthma prevalence. Most deaths occur before hospital admission.

7.1. Patients at risk of asthma-related cardiac arrest

The risk of near-fatal asthma attacks is not necessarily related to asthma severity.

Patients most at risk include those with:

- a history of near-fatal asthma requiring intubation and mechanical ventilation
- a hospitalisation or emergency care for asthma in the past year
- low or no use of inhaled corticosteroids
- an increasing use and dependence of beta-2 agonists
- anxiety, depressive disorders and/or poor compliance with therapy
- food allergy in a patient with asthma

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7.2. Causes of cardiac arrest

Cardiac arrest in the asthmatic is often a terminal event after a period of hypoxaemia; occasionally, it may be sudden. Cardiac arrest in asthmatics has been linked to:

- Severe bronchospasm and mucous plugging leading to asphyxia (most frequent cause of death).
- Cardiac arrhythmias due to hypoxia, stimulant drugs (e.g. ß-adrenergic agonists, aminophylline) or electrolyte abnormalities.
- Dynamic hyperinflation, i.e. auto-positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Auto-PEEP is caused by air trapping and 'breath stacking' (air entering the lungs and being unable to escape). Gradual build-up of pressure occurs and reduces venous return and blood pressure.
- Tension pneumothorax (often bilateral).

The 4 Hs and 4 Ts approach to reversible causes will help identify these causes in cardiac arrest.

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7.3. Assessment and treatment

Use the ABCDE approach to assess severity and guide treatment. The severity of acute asthma is summarised in table 12.2.

Asthma severity		
Near-fatal asthma	Raised Pa_{CO_2} and/or requiring mechanical ventilation with raised inflation pressures	
Life-threatening asthma	Any one of the following signs:	
	Clinical signs	**Measurements**
	Altered conscious level	PEF < 33 % best or predicted
	Exhaustion	Sp_{O_2} < 92 %
	Arrhythmia	Pa_{O_2} < 8 kPa
	Hypotension	'normal' Pa_{CO_2} (4.6-6.0 kPa)
	Cyanosis	
	Silent chest	
	Poor expiratory effort	
Acute severe asthma	Any one of: - PEF 33-50 % best or predicted - respiratory rate ≥ 25 min^{-1} - heart rate ≥ 110 min^{-1} - inability to complete sentences in one breath	

PEF = peak expiratory flow

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- Wheezing is a common physical finding, but severity does not correlate with the degree of airway obstruction. Other causes of wheezing include: pulmonary oedema, chronic obstructive pulmonary disease (COPD), pneumonia, anaphylaxia, foreign bodies, pulmonary embolism, subglottic mass.
- The patient with acute severe asthma requires aggressive medical management to prevent deterioration. Experienced clinicians should treat these patients in a critical care area. Patients with Sp_{O_2} < 92 % or with features of life-threatening asthma are at risk of hypercapnia and require arterial blood gas measurement.
- Use a concentration of inspired oxygen that will achieve an Sp_{O_2} 94-98 %. High-flow oxygen by mask is sometimes necessary. Lack of pulse oximetry should not prevent the use of oxygen.
- Salbutamol (5 mg nebulised) is the main therapy for acute asthma. Repeated doses every 15-20 min, or continuous doses, may be needed. Nebuliser units that can be driven by high-flow oxygen (at least 6 l min^{-1}) should be used. Remember that

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nebulised drugs will not be delivered to the lungs effectively if the patient is tired and hypoventilating. If a nebuliser is not immediately available beta-2 agonists can be temporarily administered by repeating activations of a metered dose inhaler via a large volume spacer device.

- Nebulised anticholinergics (ipratropium 0.5 mg 4-6 hourly) may produce additional bronchodilation in severe asthma and in those who do not respond to beta-agonists.
- Inhaled magnesium sulphate is currently not recommended for the treatment of acute asthma.
- Intravenous magnesium sulphate (2 g IV slowly = 8 mmol) may be useful in patients with acute severe asthma (PEF < 50% best or predicted) who have not had a good initial response to inhaled bronchodilator therapy. The most commonly reported adverse effects are flushing, fatigue, nausea, headache and hypotension.
- Early use of systemic corticosteroids for acute asthma significantly reduces hospital admission rates. Although there is no difference in clinical effects between oral and IV formulations of corticosteroids, the IV route is preferable because patients with near-fatal asthma may vomit or be unable to swallow.
- Consider intravenous salbutamol in patients unresponsive to nebulised therapy or where nebulised/inhaled therapy is not possible (e.g. a patient receiving bag-mask ventilation). Give as either a slow IV injection (250 mcg IV slowly) or continuous infusion of 3-20 mcg min^{-1}.
- There is no evidence of benefit and a higher incidence of adverse effects for intravenous aminophylline compared with standard care alone. If after obtaining senior advice the decision is taken to administer IV aminophylline a loading dose of 5 mg kg^{-1} is given over 20-30 min (unless on maintenance therapy), followed by an infusion of 500-700 mcg $kg^{-1}h^{-1}$. Serum theophylline concentrations should be maintained below 20 mcg ml^{-1} to avoid toxicity.

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Cardiac arrest

- Follow standard BLS and ALS protocols. Ventilation will be difficult because of increased airway resistance; try to avoid gastric inflation.
- Intubate the trachea early. There is a significant risk of gastric inflation and hypoventilation of the lungs when attempting to ventilate a severe asthmatic without a tracheal tube.
- Respiratory rates of 8-10 breaths per minute and a tidal volume required for a normal chest rise during CPR should minimise dynamic hyperinflation of the lungs (air trapping).
- If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest wall and/or a period of apnoea (disconnection of tracheal tube) may relieve gas-trapping. Although this procedure is supported by limited evidence, it is unlikely to be harmful in an otherwise desperate situation.
- Dynamic hyperinflation increases transthoracic impedance, but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with a higher impedance. As with standard ALS defibrillation protocols, consider increasing defibrillation energy if the first shock is unsuccessful.
- Look for reversible causes using the 4 Hs and 4 Ts approach.
- Tension pneumothorax can be difficult to diagnose in cardiac arrest; it may be indicated by unilateral expansion of the chest wall, shifting of the trachea, and subcutaneous emphysema. Pleural ultrasound in skilled hands is faster and more sensitive than chest X-ray for the detection of pneumothorax. Early needle decompression (thoracocentesis) followed by chest drain insertion is needed. Needle decompression may fail due to inadequate needle length. In the ventilated patient, thoracostomy (a surgical hole in the chest wall and pleura) may be quicker to do and more effective for decompressing the pneumothorax *(see trauma section)*.
- Always consider bilateral pneumothoraces in asthma-related cardiac arrest.

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