



EDITORIALS

Improving treatment of asthma attacks in children

UK children and young people have the highest death rate from asthma in Europe

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An acute asthma attack is frightening and serious. The common term for these attacks—"exacerbation," defined as the worsening of a disease or an increase in its symptoms—undervalues an event that is predictive of further acute deterioration and death, with serious long term consequences, including restriction of lung growth into adulthood.¹ Asthma still kills people of all ages²; there are preventable factors in the majority of deaths, and the UK has the highest death rate from asthma in children and young people in Europe.³ We must do better.

The underlying pathophysiology of an asthma attack is severe airway obstruction as a result of smooth muscle constriction and plugging of the airway by inflammatory cells and mucus. This leads to increased work of breathing and also hypoxaemia because of ventilation: perfusion (V:Q) mismatch. The signs of airway narrowing (wheeze) and increased work of breathing (subcostal recession, use of accessory muscles) are readily apparent and are often the focus of treatment, but hypoxaemia may be difficult to detect clinically, at least initially, and is thus easily forgotten.

The mainstay of treatment is β_2 agonists. These were traditionally delivered by nebuliser until overwhelming evidence showed that spacers were equally if not more effective in mild to moderate attacks,⁴ and (particularly relevant to low and middle income settings) homemade spacers were just as good as commercial ones.⁵ Spacers are now used routinely in emergency departments, but primary care has been slower to move away from nebulisers, which can cause harm, particularly when driven by air.

When treating an asthma attack, it is important to remember the harms as well as the benefits of short acting β_2 agonists. However delivered, inhaled β_2 agonists are absorbed from the bronchial tree, the oropharynx, and possibly the upper gastrointestinal tract into the systemic circulation. The response to alveolar hypoxia is vasoconstriction, and systemic administration or absorption of β_2 agonists may reverse this, potentially worsening V:Q mismatch and hypoxaemia. Treatment of a severe attack must include both β_2 agonists and oxygen, so nebulisers driven by air may worsen hypoxaemia. More rarely, even oxygen driven nebulisers may have the same effect in children with focal consolidation.⁶

The nebuliser is not yet ready for the therapeutic junk heap, however. In very severe asthma, continuous nebulised salbutamol, sometimes with an intravenous loading dose and always with oxygen administration, is a valuable strategy for patients being closely monitored in high dependency settings.⁷ It is also reasonable to use an oxygen driven nebuliser for any severe attack in primary care, but only while waiting for emergency services.

The appropriate choice of delivery device in either primary or secondary care depends on accurate differentiation of severe attacks from the rest—and a failure to recognise a life threatening attack is a recurring theme in national reviews of asthma deaths.² The UK and Global Initiative for Asthma (GINA) guidelines both have helpful tables that define the population of patients at risk and the clinical findings associated with a severe attack.^{8,9} The clinician must track symptoms and signs in response to treatment, and assessment should include pulse oximetry.

Assessing patients without objectively measuring oxygen saturation is unacceptable in any setting given that pulse oximeters can be obtained cheaply and easily with probes appropriate for every age; that cyanosis may be difficult to detect, especially in a poorly lit room in the community; and that hypoxaemia can be lethal. There is a theoretical risk that uncontrolled high flow oxygen can lead to serious hypercapnia even in young adults with asthma.¹⁰ But for short term treatment in primary care, where there may be no ability to titrate oxygen, it is far better to err on the side of caution and correct hypoxaemia without worrying about hypercapnia.

In summary, the age old message remains that doctors must assess patients with an asthma attack using objective measurements, administer β_2 agonists with a device appropriate to severity, and monitor the response to treatment. In primary care, nebulisers should be reserved for severe or life threatening attacks, as a temporary rescue strategy before transfer to hospital. Importantly, an asthma attack must trigger a focused strategy to reduce the risk of further attacks, including specialist referral if attacks are frequent. Clinicians, patients, and family members should review what went wrong, whether treatment should be modified, and how best to prevent further events. It is of course

essential to stop people dying from acute asthma attacks, but we as a community need to go to the next level, move from reaction to prevention, and make asthma attacks a real never event.¹¹

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