

Prolonged hypoxaemia after nebulised salbutamol

Gary Connett, Warren Lenney

Abstract

Pulse oximetry is increasingly used to assess hypoxaemia in respiratory illnesses. Six children presenting with acute asthma and prolonged falls in oxygen saturation values after treatment with salbutamol are described who were subsequently shown to have pneumonic consolidation on chest radiography.

(Thorax 1993;48:574-575)

Arterial oxygen desaturation after bronchodilator therapy is well recognised in acute asthma and infants with bronchiolitis.^{1,2} Typically the maximum fall in saturation occurs within the first 5-10 minutes after treatment and is self limiting within 15 minutes. The importance of continued oxygen treatment after nebulisation has been emphasised.³ We describe six children with acute asthma who showed significant falls in oxygen saturation for up to one hour after treatment with salbutamol.

Royal Alexandra
Hospital for Sick
Children, Dyke Road,
Brighton BN1 3JN

G Connett
W Lenney

Reprint requests to:
Dr W Lenney

Received 10 April 1992

Returned to authors

22 June 1992

Revised version received

1 July 1992

Accepted 9 July 1992

Methods

Over a six month period, children over 18 months referred to the Royal Alexandra Hospital for Sick Children, Brighton with acute asthma had oxygen saturation measurements recorded before and at two, five, and 10 minutes after administration of 5 mg nebulised salbutamol (1 ml diluted in 2 ml saline). If the 10 minute measurement was at

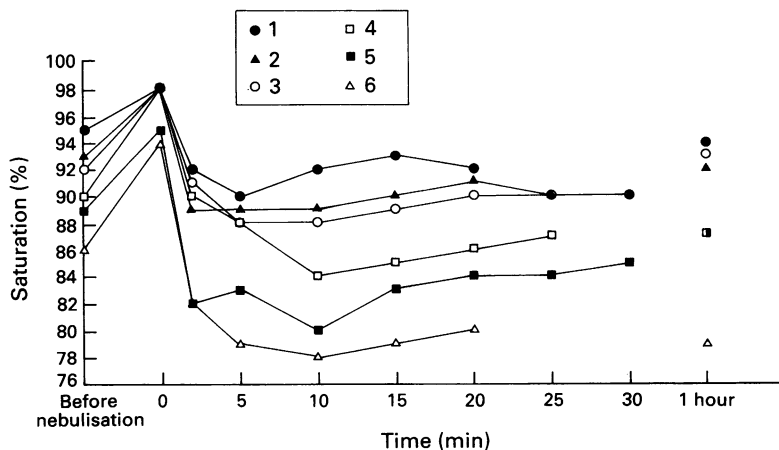
least 2% below the initial saturation value, further measurements were obtained over the next hour. Nebulisers were driven by oxygen at 8 l/min and saturation levels were measured with a Nellcor N200 pulse oximeter. Individual measurements were recorded at specified times to ensure that a good quality pulse signal was present. In addition, heart rate, respiratory rate, peak flow (when possible), and a clinical assessment of recession and accessory muscle use were recorded before and at least 10 minutes after the nebuliser. The use of oxygen in children after nebulisation was based on clinical assessment irrespective of saturation values. Children in whom oxygen was thought to be indicated received sufficient flow rates to achieve saturations above 90% until a final saturation measurement in air was recorded one hour after the nebuliser. All assessments were made by the same observer (GC), depending on his availability to see each referral within half an hour of arrival.

Results

Of 111 children examined, six were identified as having oxygen saturation values at least 2% below prenebuliser values up to 20 minutes after treatment (fig 1). All had suffered previous episodes of wheezing and were not distinguishable from other wheezy children on presentation at the hospital. In cases 4, 5, and 6, falls in oxygen saturation were to levels at which oxygen therapy was thought to be necessary. Despite increased hypoxaemia there was no clinical evidence of increased airways obstruction in any of the children when reassessed 10 minutes after receiving nebulised salbutamol (table). Chest radiographs were obtained and all showed obvious pneumonic changes. There were either areas of lobar consolidation or dense infiltrates scattered throughout the lung fields. Some had additional areas of atelectasis. Viral serological tests identified adenovirus in two and mycoplasma in one patient. All showed a slow response to inpatient treatment despite the use of corticosteroids. Three children have subsequently been readmitted with acute asthma after resolution of the previous radiographic changes and none had prolonged falls in oxygen saturation levels after salbutamol on these occasions.

Discussion

A measurable fall in saturation value of $\geq 2\%$ is likely to represent a significant change in arterial oxygen saturation.⁴ Although uncommon, this study reports that such falls can occur for prolonged periods after salbutamol without evidence of worsening bronchoconstriction. These children would not have undergone chest radiography had they been admitted routinely, and their lung consolidation was not apparent clinically. Although the clinical presentation in this group of children was similar to that of other patients with asthma, their radiographs were not typical of



Oxygen saturation values before and up to one hour after nebulised salbutamol in six children admitted with acute asthma.

Clinical details before and 10 minutes after administration of salbutamol.

Patient no	Age (years)	Heart rate (beats/min)		Respiratory rate (breaths/min)		PEF (% of best)		Recession	
		Before	After	Before	After	Before	After	Before	After
1	2.1	168	160	68	54	—	—	++	+
2	6.2	154	138	58	60	30	35	++++	+++
3	1.8	148	152	64	56	—	—	+++	++
4	5.0	130	148	40	40	30	42	+	—
5	2.5	152	140	60	50	—	—	++	+
6	1.6	178	168	76	68	—	—	++++	+++

PEF—peak expiratory flow; recession = +—moderate; ++—severe; +++—with use of accessory muscles; ++++—significant use of accessory muscles.

the radiographic abnormalities described in acute asthma,⁵ and their subsequent recoveries were prolonged.

Hypoxaemia after treatment with bronchodilators is thought to result from worsening ventilation perfusion mismatch. These abnormalities in gas exchange have been shown with inert gas elimination methods.⁵ Hypoxaemia in asthma is caused by the perfusion of poorly ventilated lung units. After salbutamol, changes in vascular tone sometimes precede bronchodilatation and increase mismatch before the compensatory effects of increased cardiac output and decreased bronchomotor tone restore or improve oxygenation.⁶ Studies in severe asthmatic patients reveal that hypoxaemia is also caused by ventilation perfusion mismatch in these cases, and true shunting (the perfusion of non-ventilated lung areas) is not a feature of acute asthma because of the extensive collateral ventilation at the bronchiolar level.⁷ These studies have excluded patients with radiographic evidence of lung consolidation. Such cases might have small shunts of pulmonary blood through these non-ventilated lung areas. The vasodilator effects of salbutamol can overcome hypoxic pulmonary vasoconstriction and this would cause increased perfusion of consolidated areas. Such an increase in shunting could have caused the prolonged

falls in saturation identified in our six children.

Pulse oximeters have greatly facilitated the assessment of hypoxaemia in acute asthma. Physicians should consider the presence of pneumonic consolidation complicating asthma in those cases with prolonged falls in saturation despite improvements in airways obstruction.

We thank Dr Evans and Dr Trounce for allowing us to study their patients. GC is supported by the Royal Alexandra Hospital Rocking Horse Appeal.

- 1 Gleeson JGA, Green S, Price JF. Air or oxygen as driving gas for nebulised salbutamol. *Arch Dis Child* 1988;63:900-4.
- 2 Ling Ho, Collis G, Landau LI, Le Soeuf PN. Effect of salbutamol on oxygen saturation in bronchiolitis. *Arch Dis Child* 1991;66:1061-4.
- 3 Tal A, Pasterkamp H, Leahy F. Arterial oxygen desaturation following salbutamol inhalation in acute asthma. *Chest* 1984;86:868-9.
- 4 Nickerson BG, Sarkisian C, Tremper K. Bias and precision of pulse oximeters and arterial oximeters. *Chest* 1988;93:515-7.
- 5 Eggleston PA, Ward BH, Pierson WE, Bierman CW. Radiographic abnormalities in acute asthma in children. *Pediatrics* 1974;54:442-9.
- 6 Wagner PD, Dantzker DR, Iacovoni VE, Tomlin WC, West JB. Ventilation-perfusion inequality in asymptomatic asthma. *Am Rev Respir Dis* 1978;118:511-24.
- 7 Rodriguez-Roisin R, Ballester E, Roca J, Torres A, Wagner PD. Mechanisms of hypoxaemia in patients with status asthmaticus requiring mechanical ventilation. *Am Rev Respir Dis* 1989;139:732-9.