

Arterial blood gas analysis or oxygen saturation in the assessment of acute asthma?

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Abstract

Background – A study was undertaken to determine if arterial blood gas estimation is always necessary in the assessment of patients presenting to hospital with acute severe asthma, or whether oxygen saturation as measured by pulse oximetry is a reliable screening test for predicting those in respiratory failure.

Methods – A prospective study was conducted in a specialist respiratory medical unit. Arterial blood gas tensions and pulse oximetry were measured in 89 consecutive patients admitted with acute severe asthma. Respiratory failure was defined as $\text{PaO}_2 < 8.0 \text{ kPa}$ or $\text{PaCO}_2 > 6 \text{ kPa}$.

Results – When oxygen saturation was 92% or higher (72 patients) respiratory failure was found in three (4.2%) cases. In the 82 patients with a saturation of 90% or higher six patients (7.3%) had respiratory failure.

Conclusions – In the initial assessment of acute severe asthma an oxygen saturation of >92% suggests that respiratory failure is unlikely and therefore arterial blood gas measurement is unnecessary. This study is only relevant to the assessment of asthmatic patients at presentation. Other parameters of severity must be continually assessed in all asthmatic patients admitted to hospital irrespective of the initial SaO_2 , and blood gases measured when clinically indicated.

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Keywords: asthma, oximetry, arterial gases.

Hypoxia is a universal finding in acute exacerbations of asthma but respiratory failure with an arterial oxygen tension (PaO_2) below 8 kPa or a raised arterial carbon dioxide tension (PaCO_2) above 6 kPa occurs in no more than a third of patients.^{1,2} Cyanosis is a late sign of respiratory failure in severe asthma and indicates a very severe life threatening attack.³ Clinical features of less pronounced respiratory failure are unreliable and therefore guidelines recommend that arterial blood gas tensions should be measured on all patients presenting to hospital with acute severe asthma.³

Arterial puncture can be painful and may lead to an increase in patients' anxiety. Bruising and haematomas are not unknown and are unpleasant. For the house physician sampling is time consuming and can result in needle stick injury if due care is not taken.

We therefore aimed to determine whether arterial blood gas estimation was necessary in

all patients presenting with acute severe asthma or whether oxygen saturation (SaO_2) as measured by pulse oximetry was a safe and reliable alternative in predicting those in respiratory failure and therefore in need of more intensive management. We also aimed to determine the SaO_2 value below which arterial gases should be taken for the safe management of asthma.

Methods

Arterial blood gas tensions were measured using the Coral Pulsator single use heparinised arterial blood sampling system and a Radiometer ABL30 acid-base analyser and oxygen saturation via an ear or finger probe⁴ attached to an Ohmeda Biox 3740 pulse oximeter in 89 consecutive admissions between July 1992 and April 1993 to our respiratory unit with acute severe asthma. Both measurements were made as part of the initial clinical assessment. No attempt was made to make measurements with the patients breathing air or a standard concentration of oxygen since this study was undertaken on patients being managed according to recommended guidelines.⁵

As markers of the severity of asthma on admission and the degree of reversibility, peak expiratory flow (PEF) on admission was expressed as a percentage of the patient's best PEF value previously recorded out of hospital or, when these data were not available, their maximum PEF value prior to discharge.

Results

The age range and smoking history of the 89 patients are shown in table 1. The differences in age and in smoking history between those patients with SaO_2 above and below 92% were not significant. Oxygen saturation ranged between 84% and 99% (median 95%); 17 patients had $\text{SaO}_2 < 92\%$ (table 1). Eight of the 89 patients were in respiratory failure ($\text{PaO}_2 < 8 \text{ kPa}$ and/or $\text{PaCO}_2 > 6 \text{ kPa}$) (table 2). Five of the 17 patients with $\text{SaO}_2 < 92\%$ had respiratory failure. Of the 72 patients with $\text{SaO}_2 > 92\%$ three (4.2%) had arterial gas tensions indicating

Table 1 Demographic details of patients

	$\text{SaO}_2 > 92\%$ (n = 72)	$\text{SaO}_2 < 92\%$ (n = 17)
Mean (range) age (years)	32 (13-79)	43 (16-65)
Smokers (past and present)	21 (29%)	8 (47%)

SaO_2 = oxygen saturation.

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Table 2 Arterial gases and oximetry of patients in respiratory failure

Patient no.	SaO ₂ (%)	PaO ₂ (kPa)	Paco ₂ (kPa)
1	87	7.4	4.5
2	89	7.4	5
3	90	6.6	4.7
4	91	7.7	4.5
5	91	7.9	5.5
6	94	7.4	4.1
7	95	10.2	6.1
8	96	7.9	5.1

SaO₂ = oxygen saturation, PaO₂ = arterial oxygen tension, Paco₂ = arterial carbon dioxide tension.

Table 3 Summary of oximetry readings

	No. of patients	No. of patients in respiratory failure
SaO ₂ >92%	72 (80.9%)	3 (4.2%)
SaO ₂ >90%	82 (92%)	6 (7.3%)
Total	89 (100%)	8 (9.0%)

SaO₂ = oxygen saturation.

Table 4 Mean (range) clinical status of patients with respect to oxygen saturation (SaO₂) and arterial blood gas tensions (ABG)

	SaO ₂ >92%	SaO ₂ <92%	ABG not in respiratory failure	ABG in respiratory failure
Heart rate (/min)	96 (68–150)	100 (68–140)	97 (68–150)	95 (68–124)
Systolic paradox (mm Hg)	4 (0–30)	7 (0–33)	6 (0–30)	9 (0–33)
Respiratory rate (/min)	25 (15–36)	27 (20–36)	25 (15–36)	25 (20–30)
Admission PEF (l/min)	230 (60–480)	150 (60–340)	220 (60–480)	140 (75–210)
*Degree of reversibility (%)	56 (19–95)	32 (17–45)	54 (17–95)	37 (25–42)

* Admission PEF/best PEF (%).

respiratory failure (table 2). Ten patients had an SaO₂ of 90–92%, and three of them had respiratory failure (table 3). The number of patients in respiratory failure with an SaO₂ above 90% is significantly greater than those with SaO₂ above 92% ($p < 0.005$, χ^2 test).

The percentage of inspired oxygen at the time of assessment was recorded in 62 of the 89 patients (11 had an SaO₂ <92%). Forty nine (79%) had measurements recorded while breathing air (five of these had an SaO₂ <92%). Patients with an SaO₂ <92% were more likely to be on a higher percentage inspired oxygen concentration than those with an SaO₂ >92%, but this difference was not statistically significant.

Clinical measurements of the severity of the patient's asthma on admission were made in accordance with the BTS guidelines³ and are shown in table 4. Patients in respiratory failure and those with an SaO₂ <92% had lower absolute PEF values on admission and, when expressed as a percentage of their best PEF (37% and 32% respectively), than those with an SaO₂ >92% or those not in respiratory failure (56% and 54% respectively) (table 4). These values will err in underestimating the severity of the attack because the best PEF value recorded before discharge was used as the denominator for some patients and this may be less than the highest recorded as an outpatient. No patient required ventilation but eight of the 89 received intravenous aminophylline.

Discussion

These data suggest that oxygen saturation, measured by pulse oximetry, can give an indication of which patients presenting with acute severe asthma are likely to be in respiratory failure and therefore in need of more intensive management. When oxygen saturation is 92% or greater respiratory failure is unlikely. Blood gas tensions should be measured in patients with acute severe asthma whose SaO₂ is below 92% on admission regardless of their inspired oxygen concentration. Arterial sampling is not required in patients with an SaO₂ of 92% or higher unless their condition does not improve objectively or deteriorates, remembering that SaO₂ does not assess alveolar ventilation.⁶ All medical wards to which patients with asthma are admitted should be equipped with a pulse oximeter.³

This study was performed in a busy acute respiratory medical unit on asthmatic patients admitted as emergencies. Inspired oxygen concentration was not recorded in 30% of patients when SaO₂ and arterial gas tensions were measured. This is a potential weakness in the study, but is likely to approximate or even exceed routine clinical practice. Furthermore, even when recorded it may be difficult to state precisely the inspired oxygen percentage at the moment of blood gas or SaO₂ measurement. Patients may be having, or have just completed, nebulised medication driven by higher concentrations of oxygen. Inspired oxygen concentrations may have just been changed or masks may not fit properly. Although most of our measurements were taken when the patient was recorded as breathing air, any further interpretation of the data may be potentially misleading.⁷ Other factors which need to be considered when interpreting oxygen saturation measurements include a history of recent smoking and anaemia.⁸ Each will tend to give falsely high results.⁹ These data, obtained from patients shortly after their admission, suggest that an SaO₂ >92% is a clinically useful result whatever the percentage of inspired oxygen.

Admission PEF expressed as a percentage of the patient's best PEF value was lower in those patients with an SaO₂ <92% and also in those with respiratory failure. This supports the use in the British guidelines^{3,5} of a PEF value of less than 33% predicted or best to indicate a very severe life threatening attack. Heart and respiratory rates and the degree of paradox¹⁰ appear to correlate less well with severity of asthma as measured by pulse oximetry and blood gas tensions.

During the study period no patients required ventilation and recordings from such a group would have added strength to the study. The fact that two patients had SaO₂ values >92% (94% and 96%) with PaO₂ values <8 kPa was worrying but emphasises the need for continual reassessment in all patients admitted with asthma, no matter what the initial SaO₂ or PaO₂. When patients fail to improve according to objective assessment of peak flow and SaO₂, then arterial gas tensions should be measured or repeated.

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Non-tuberculous pulmonary infections in Scotland: a cluster in Lothian?

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Abstract

Background - A retrospective study was carried out to confirm the clinical impression that, in Lothian, non-tuberculous mycobacterial infections are as common as pulmonary tuberculosis.

Methods - All pulmonary isolates of *Mycobacterium tuberculosis* *bovis* and non-tuberculous mycobacteria in Scotland from April 1990 to March 1993, and the notes of all patients with *M malmoense* isolates in Lothian, were reviewed. Information on mycobacterial culture procedures in Scottish laboratories was obtained as part of an audit project.

Results - Of all pulmonary isolates of mycobacteria in Lothian 53% (108/205) were non-tuberculous strains compared with 18% (140/800) for Scotland outside Lothian. Although comparable in population size and laboratory techniques, Lothian (108) had almost twice as many isolates of non-tuberculous mycobacteria as Glasgow (56), but the proportions of *M malmoense* and *M avium intracellulare* complex were similar in both areas. Of 41 patients with *M malmoense* isolates in Lothian 30 (75%) had clinically significant lung disease; only one was HIV positive.

Conclusions - Non-tuberculous mycobacteria pose an increasing clinical problem in Scotland as a cause of pulmonary disease. There is a cluster of cases with *M malmoense* infection in Lothian which cannot be attributed to the high local prevalence of HIV.

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Keywords: non-tuberculous mycobacteria, *Mycobacterium malmoense*, Lothian.

A review of all pulmonary mycobacterial isolates in Scotland over three years revealed that

almost half of all Scottish isolates of *Mycobacterium malmoense* were from patients in Lothian. *M malmoense* was first described as a pulmonary pathogen in 1977¹ but has since been reported in increasing frequency in Europe.²⁻⁷ The organism has only rarely been isolated in the USA and from immunocompromised patients.⁸⁻¹⁰ This study attempted to determine whether the clustering of patients with *M malmoense* in Lothian was genuine or due to the use of different laboratory methods.

Methods

The names of all patients in Scotland with positive pulmonary isolates (sputum, bronchial washings, lung tissue, pleural fluid, and biopsies) of *M tuberculosis*, *M bovis*, and non-tuberculous mycobacteria during the period April 1990 to March 1993 were obtained from the Scottish Mycobacteria Reference Laboratory in Edinburgh. Age, sex, postcode, HIV status, and details of antimycobacterial treatment for these patients were collected from general practitioners and consultant colleagues. Information on mycobacterial culture procedures in Scottish bacteriology laboratories was available through an audit project of the Reference Laboratory. In order to assess possible clustering of cases within Lothian we related the number of patients with *M malmoense* strains to their postcode districts and the population figure of that area based on the 1991 census.

Results

All data concerning positive mycobacterial cultures represent pulmonary samples of patients which were received by the laboratories during the period April 1990 to March 1993.

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