Chronic stable asthma and the normal arterial pressure of carbon dioxide in hypoxia

G M COCHRANE, J G PRIOR, C B WOLFF

Summary and conclusions

Arterial blood-gas tensions, pH, and peak expiratory flow rate were measured in 29 patients with chronic asthma in a stable state. The hypoxia in these patients was found to be comparable with the hypoxia seen in normal subjects at high altitude in its effects on arterial pressure of carbon dioxide (Paco₂). These results suggest that in patients with asthma the Paco₂, taken as normal should be related to the arterial oxygen tension. Any increase in the observed value compared with this predicted value indicates impaired respiratory control. This may well help in assessing the patients at greatest risk during an attack of asthma.

References


(Accepted 18 July 1980)
All patients had extrinsic asthma. We excluded those with clinical features suggesting chronic obstructive bronchitis. Most patients were studied at a routine follow-up asthma clinic; a few were studied on admission to hospital for investigation or treatment of chronic intractable asthma. Criteria for chronicity were based mainly on history and a normal arterial pH. In a few patients peak expiratory flow rate was known to have been stable over the preceding week.

Arterial blood samples were obtained by percutaneous puncture of the non-dominant radial artery. Arterial blood was analysed for pH, PaO₂, and PaCO₂ with an IL 213 blood-gas analyser. Calibrations for pH were made using standard buffers (N B S Washington) and for PaCO₂ and PaO₂ using known gas mixtures previously analysed with the Haldane apparatus. Patients were excluded from the study if their arterial pH was outside the range 7.35-7.46, as this suggested that the asthma was not chronic. Peak expiratory flow rate was measured within a few minutes of arterial puncture.

**Results**

Twenty-nine patients with extrinsic asthma (21 men and eight women), whose ages ranged between 19 and 44 years, were included in the analysis. Peak expiratory flow rate varied between 100 and 490 l/min. The table shows arterial blood-gas tensions, arterial pH, and peak expiratory flow rate for each patient.

**Arterial pressures of oxygen (PaO₂) and carbon dioxide (PaCO₂), pH values, and peak expiratory flow rate in patients with chronic stable asthma**

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**Inpatients**

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*Values recorded in same patient during two separate admissions. Mean arterial pH was 7.423 ± 0.003, mean peak expiratory flow rate 254 l/min.

Conversion: SI to traditional units—PaCO₂ and PaO₂: 1 kPa = 7.5 mm Hg.

Figure 1 shows the relation between PaCO₂ and PaO₂. The trend towards a lower PaCO₂ accompanying a low PaO₂ is highly significant (p<0.001). The relation is: PaCO₂ predicted = 0.23 PaO₂ + 2.2 kPa (or PaCO₂ predicted = 0.23 PaO₂ + 16.6 mm Hg). This is not significantly different from the relation given by Wolff۷ for normal subjects acclimatised to altitude hypoxia.

**Discussion**

Asthma is defined as reversible airflow obstruction. Despite considerable variation in the degree of obstruction, however, many patients maintain remarkably stable arterial blood-gas tensions. Often, despite improvement in peak expiratory flow rate after admission to hospital, the arterial blood-gas tensions fail to improve for several days.۷ Reubuck and Read۸ showed that the mechanism leading to the fall in PaO₂ is only partially related to peak expiratory flow rate. Reduction in PaO₂ is associated with plugging of small airways, leading to a shunt effect.۷۴ Persistence of small-airway dysfunction, with associated hypoxia but few symptoms, is well documented in asthma.۷

We studied arterial blood-gas tensions in patients with chronic stable asthma to investigate whether there is a relation between PaO₂ and PaCO₂ similar to that found in normal subjects acclimatised to altitude hypoxia.
Changes in glycosylated haemoglobin after poor control in insulin-dependent diabetics

ANDREW P BROOKS, ISOBEL M NAIRN, JOYCE D BAIRD

Summary and conclusions

Glycosylated haemoglobin (HbA1c) was measured in seven insulin-dependent diabetic patients before, during, and after a seven-day period of monitored poor control. There was considerable individual variation in the pattern and degree of change in HbA1c concentration induced by poor control and the time when it occurred. Greater increases in HbA1c were seen during the period of metabolic derangement than in the subsequent two months.

More information is required before HbA1c estimations are widely used clinically to monitor control in individual diabetics.

Introduction

Concentrations of glycosylated haemoglobin (HbA1c) are raised in diabetic patients and, since the glucose linkage is considered to be relatively stable, are thought to reflect the mean blood glucose concentration during the preceding one to two months. Hence HbA1c estimations are used as a means of monitoring the degree of overall control of blood glucose achieved in individual diabetics. There are, however, many problems associated with measuring HbA1c and neither the time relation between changes in blood glucose and HbA1c concentrations nor the stability of HbA1c nor its sensitivity in detecting poor control has been clearly defined.

We compared two common methods of estimating HbA1c and examined the change in concentration of HbA1c resulting from a period of poor control deliberately induced in long-standing, insulin-treated diabetic patients maintained under strictly monitored, metabolic conditions.

Patients and methods

One male and six female diabetic patients being treated with insulin were studied (table I). All were fully ambulant, none was obese, and none had retinopathy, neuropathy, or nephropathy. All gave informed consent to the investigation.

The study covered 13 weeks and was divided into three periods. In the initial three-week assessment period patients were at home following their usual diet and insulin regimen and undertaking normal activity. They were visited each week on the morning after completing a 24-hour urine collection for measuring urinary glucose excretion, and fasting blood was taken for estimating whole blood true glucose, serum lipid, and HbA1c concentrations.

References

1 Wolff CB. Normal respiration in chronic hypoxia. J Physiol 1980;283:118-9P.

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