

Interpretation of Arterial Carbon Dioxide Tension in Laryngotracheobronchitis

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SUMMARY

The arterial carbon dioxide tension ($p_a\text{CO}_2$) of 57 patients with laryngotracheobronchitis was matched to the degree of obstruction as assessed by clinical methods.

A constant relationship between $p_a\text{CO}_2$ and the degree of obstruction was observed. The greatest obstruction was accompanied by the highest $p_a\text{CO}_2$ and vice versa. However, with the least obstruction patients hyperventilated. The rise in $p_a\text{CO}_2$ that accompanied increasing obstruction is relative to the initial hyperventilatory level. Therefore absolute hypoventilation was seldom present when obstruction had become significant.

S. Afr. Med. J., **48**, 1152 (1974).

In some respiratory disorders correlation of the degree of airway obstruction with the alteration in arterial carbon dioxide tension ($p_a\text{CO}_2$) has been observed, and is useful in patient management.¹⁻³ In laryngotracheobronchitis (LTB), however, this relationship has not been noted; general opinion has indeed been that the $p_a\text{CO}_2$ in LTB

is not helpful in assessment,⁴⁻⁷ or that it can remain normal until immediately before acute cardiorespiratory failure.⁵

From a study of blood gases in LTB,⁸ facets of the $p_a\text{CO}_2$ are presented. It is contended that the $p_a\text{CO}_2$ does behave in a predictable manner and can be useful in judging the degree of upper airway obstruction (UAO).

PATIENTS AND METHODS

Fifty-seven Black children with LTB, aged 5 months to 5 years (median 16 months), were studied. The aetiology was viral in all cases, 75% being related to measles. Included were patients with all degrees of UAO. The clinical features which have been related to $p_a\text{CO}_2$ are indicated under 'Results'.

The laboratory methods of analysis of arterial blood samples have been previously detailed.¹⁰ The normal laboratory value for $p_a\text{CO}_2$ in this age group is 37 ± 4 mmHg.

RESULTS

$p_a\text{CO}_2$ and Severity of UAO (Table I)

The severity of upper airway obstruction (UAO) was defined clinically in the following manner:

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Date received: 22 January 1974.

- (a) mild—stridor with intercostal and subcostal recession;
- (b) moderate—as (a) with reduced breath sounds on auscultation.
- (c) severe—as (b), with cyanosis and/or signs of fatigue, i.e., muscular hypotonia or stupor.

Patients were classified into one of these three groups, and blood gas analyses done; the mean $p_a\text{CO}_2$ of each group appears in Table I.

TABLE I. SEVERITY OF UAO RELATED TO $p_a\text{CO}_2$

| Grade of UAO | $p_a\text{CO}_2$ (mmHg) | | |
|--------------|-------------------------|----|-----|
| | Mean | SD | No. |
| Severe | 41 | 12 | 13 |
| Moderate | 39* | 10 | 30 |
| Mild | 32* | 6 | 14 |

* $P < 0.01$

The $p_a\text{CO}_2$ of the moderate and mild groups was significantly different ($P < 0.01$). That of the mild group was significantly below normal ($P < 0.05$).

$p_a\text{CO}_2$ and Air Entry on Auscultation (Fig. 1)

In UAO breath sounds are often less easily audible on auscultation. The degree to which air entry was impaired was described as reduced (grade I), and almost inaudible (grade II).

Patients were regrouped according to their air entry and the mean $p_a\text{CO}_2$ of each group was calculated (Fig. 1). Patients with normal air entry had a mean $p_a\text{CO}_2$ of 31 ± 6 mmHg, which was significantly below those with grade I reduction in air entry (39 ± 9 mmHg; $P < 0.01$), and also below the normal level ($P < 0.01$).

$p_a\text{CO}_2$ Before and After Intubation (Table II)

In those children who required tracheal intubation to relieve their UAO, the decision was taken on clinical grounds alone, and all the severe and half of the moderately classified patients were intubated.

Arterial blood gases were measured in 24 of these children before and 30 minutes after intubation. In almost all patients there was a fall of $p_a\text{CO}_2$ after intubation: the mean of the differences was significant ($P < 0.01$).

TABLE II. EFFECT OF INTUBATION ON $p_a\text{CO}_2$

| | $p_a\text{CO}_2$ (mmHg) |
|--------------------------|-------------------------|
| Before intubation (mean) | 40 ± 12 |
| After intubation (mean) | 33 ± 10 |
| Mean of differences | -7 ± 11 |
| P value | < 0.01 |
| No. of patients | 24 |

DISCUSSION

The estimation of degree of airway obstruction was by clinical means in this study. Nevertheless, from 3 observations, the $p_a\text{CO}_2$ did appear to reflect the severity of obstruction: when the obstruction was least the $p_a\text{CO}_2$ was low, and when greater the $p_a\text{CO}_2$ was higher.

In patients who required intubation there was no doubt on clinical criteria that an obstruction had been relieved, as evidenced by an increased air entry on auscultation, by reduction of respiratory effort and by the patient usually falling asleep. These events were accompanied by a significant fall in $p_a\text{CO}_2$ (Table II). This hyperventilation could not be attributed to the presence of an endotracheal tube, for a similarly low $p_a\text{CO}_2$ existed in mild (non-intubated) patients (32 ± 6 mmHg). It is reasonable to suppose that the calibre of an endotracheal tube is less than that of a normal larynx and would represent a small degree of obstruction, perhaps much the same as that of patients in the clinically mild group. Thus, both in the postintubation state and in mild LTB, a minor degree of obstruction resulted in a similar $p_a\text{CO}_2$, significantly below normal levels. Thus mild laryngeal obstruction was accompanied by hyperventilation.

Reduced air entry on auscultation is an accepted physical sign of airway obstruction. Grading of the degree of alteration must be observer-dependent, and open to criticism. However, as it was done by one observer, error was minimised. Further, the identification of patients with normal and those with almost inaudible air entry was simple. The remaining patients were grouped together.

Varying degrees of air entry were present in all severity groups of LTB except the mild, where it was normal in all cases. When patients were reclassified according to air entry, the mean $p_a\text{CO}_2$ rose as the impairment increased (Fig. 1). Those with grade I air entry reduction had a mean $p_a\text{CO}_2$ that was higher than those with normal air entry ($P < 0.01$), but the difference from those with grade II reduction was not significant. Further, the $p_a\text{CO}_2$ of patients with normal air entry was significantly less than normal ($P < 0.01$).

It is possible that infants react differently to UAO compared with older children. This was tested by dividing each severity group into patients over and under 1 year of age. No difference in mean $p_a\text{CO}_2$ of the two age groups was noted.

Thus observations of patients with various degrees of UAO suggested that $p_a\text{CO}_2$ did alter in a predictable manner. The least obstruction caused a $p_a\text{CO}_2$ below normal, after which it rose with increasing obstruction. Possibly this relationship has not been stressed previously because observations on patients comprising the whole spectrum of UAO have not been considered.

With each of the three clinical methods of estimating UAO, hyperventilation accompanied mild obstruction. A subsequent rise in $p_a\text{CO}_2$ occurred with greater obstruction. The significance of this has not been recognised because the level to which the $p_a\text{CO}_2$ rose was usually within normal limits, but such a $p_a\text{CO}_2$ is in reality high for this disease.

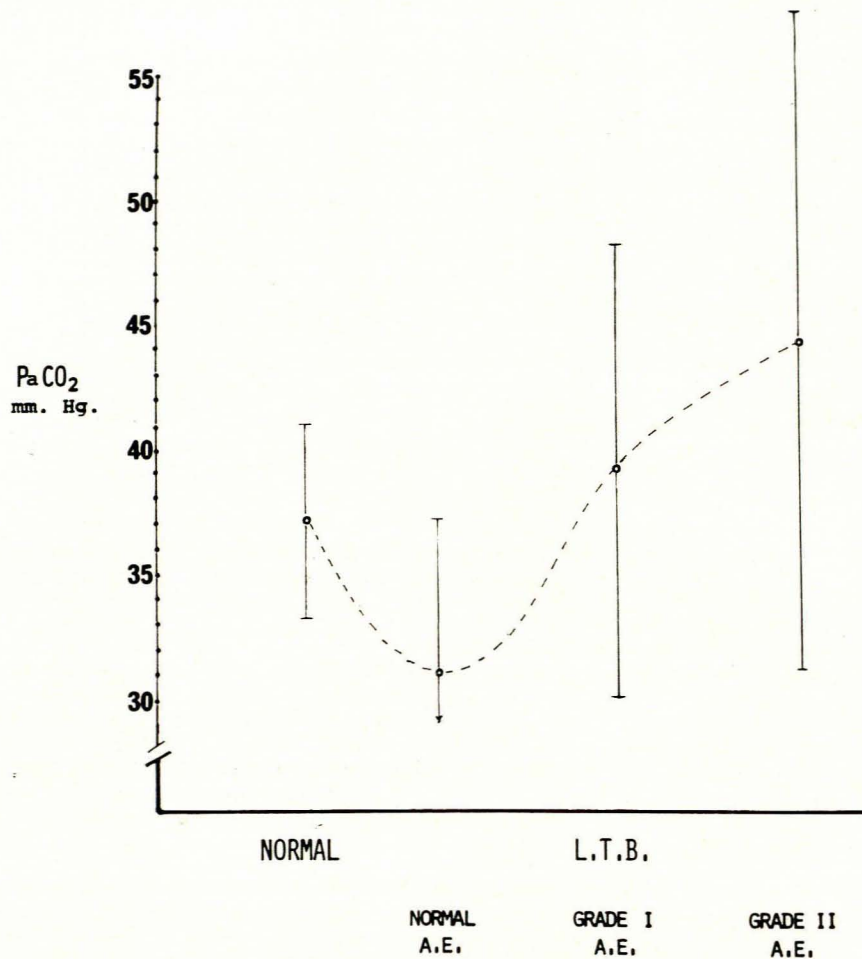


Fig. 1. Relationship of $p_a\text{CO}_2$ to air entry. The $p_a\text{CO}_2$ of patients with normal air entry (AE) was significantly less than normal ($P < 0,01$) and also less than the $p_a\text{CO}_2$ of patients with grade I air entry reduction ($P < 0,01$).

Therefore, in the presence of reduced air entry on auscultation, a favourable $p_a\text{CO}_2$ is a low one. A normal carbon dioxide tension indicates significant airway obstruction and should alert the clinician that the need for tracheal intubation is imminent.

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