Interpretation of Arterial Carbon Dioxide Tension in Laryngotracheobronchitis

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SUMMARY

The arterial carbon dioxide tension (p_aCO_2) of 57 patients with laryngotracheobronchitis was matched to the degree of obstruction as assessed by clinical methods.

A constant relationship between p_aCO_2 and the degree of obstruction was observed. The greatest obstruction was accompanied by the highest p_aCO_2 and vice versa. However, with the least obstruction patients hyperventilated. The rise in p_aCO_2 that accompanied increasing obstruction is relative to the initial hyperventilatory level. Therefore absolute hypoventilation was seldom present when obstruction had become significant.

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In some respiratory disorders correlation of the degree of airway obstruction with the alteration in arterial carbon dioxide tension (p_aCO_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_aCO_2 in LTB is not helpful in assessment,[4-7] or that it can remain normal until immediately before acute cardiorespiratory failure.[8]

From a study of blood gases in LTB,[9] facets of the p_aCO_2 are presented. It is contended that the p_aCO_2 does behave in a predictable manner and can be useful in judging the degree of upper airway obstruction (UAO).

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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_aCO_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_aCO_2 in this age group is 37 ± 4 mmHg.

RESULTS

p_aCO_2 and Severity of UAO (Table I)

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

(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_aCO_2 of each group appears in Table I.

TABLE I. SEVERITY OF UAO RELATED TO p_aCO_2

	p_aCO_2 (mmHg)		
Grade of UAO	Mean	SD	No.
Severe	**41**	**12**	**13**
Moderate	**39***	**10**	**30**
Mild	**32***	**6**	**14**

* $P<0,01$

The p_aCO_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_aCO_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_aCO_2 of each group was calculated (Fig. 1). Patients with normal air entry had a mean p_aCO_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_aCO_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_aCO_2 after intubation: the mean of the differences was significant ($P<0,01$).

TABLE II. EFFECT OF INTUBATION ON p_aCO_2

	p_aCO_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_aCO_2 did appear to reflect the severity of obstruction: when the obstruction was least the p_aCO_2 was low, and when greater the p_aCO_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_aCO_2 (Table II). This hyperventilation could not be attributed to the presence of an endotracheal tube, for a similarly low p_aCO_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_aCO_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_aCO_2 rose as the impairment increased (Fig. 1). Those with grade I air entry reduction had a mean p_aCO_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_aCO_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_aCO_2 of the two age groups was noted.

Thus observations of patients with various degrees of UAO suggested that p_aCO_2 did alter in a predictable manner. The least obstruction caused a p_aCO_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_aCO_2 occurred with greater obstruction. The significance of this has not been recognised because the level to which the p_aCO_2 rose was usually within normal limits, but such a p_aCO_2 is in reality high for this disease.

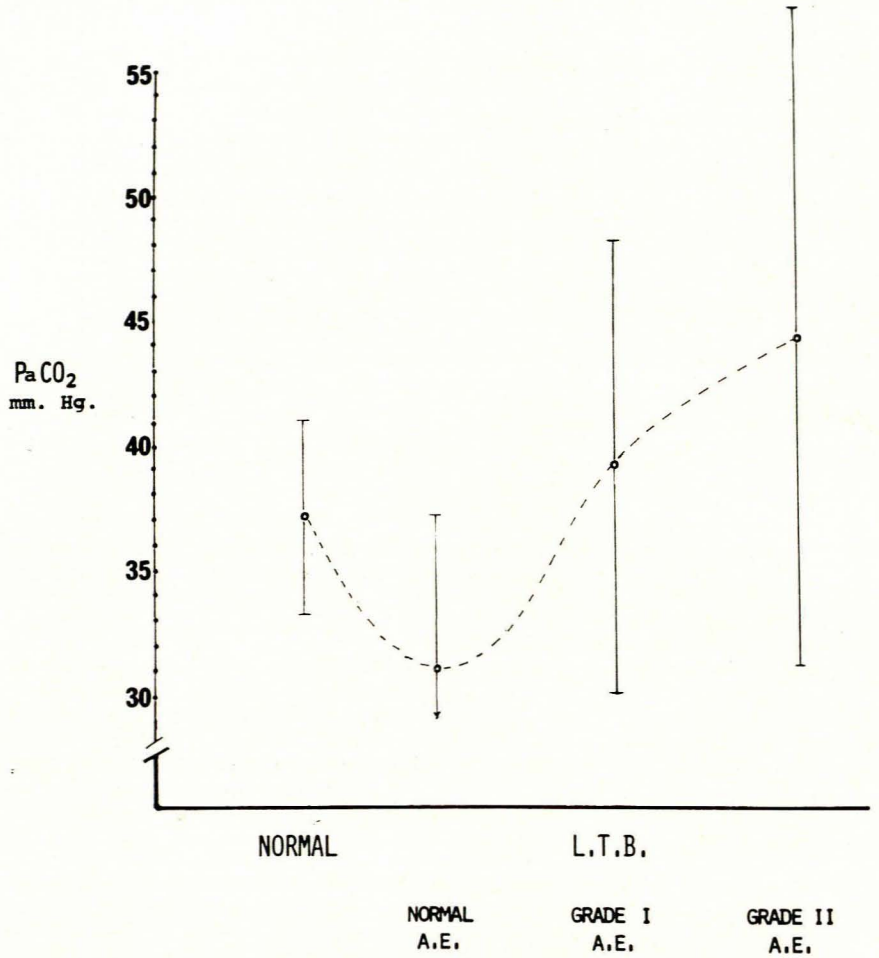

Fig. 1. Relationship of p_aCO_2 to air entry. The p_aCO_2 of patients with normal air entry (AE) was significantly less than normal ($P < 0{,}01$) and also less than the p_aCO_2 of patients with grade I air entry reduction ($P < 0{,}01$).

Therefore, in the presence of reduced air entry on auscultation, a favourable p_aCO_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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