The Acid-Base Balance of Infective Croup

A. G. WESLEY, M.B., CH.B. (CAPE TOWN), M.R.C.P. (EDIN.), D.C.H. (LOND.), Department of Paediatrics and Child Health, University of Natal and the Respiratory Unit, King Edward VIII Hospital, Durban

SUMMARY

The acid-base status of patients with upper airway obstruction secondary to viral croup, was studied over a period of 7 days. All patients, some only with the aid of intubation, alkalinized themselves. The mechanisms of this manoeuvre are discussed.


Reports of the examination of blood gases in laryngotraceobronchitis have in the past been few and cursory. This study was undertaken to follow patients from admission to hospital towards recovery, in an attempt to delineate the pattern of their response to the disease.

MATERIAL AND CLINICAL DATA

Fifty-four children, aged from 5 months to 7 years, with upper airways obstruction due to viral laryngotracheobronchitis, were studied for 7 days from admission. On admission they were graded clinically into 3 severity groups:

1. Mild cases: stridor and recession only (14 cases).
2. Severe cases: stridor and recession with indifference to stimuli and/or hypotonia and/or cyanosis (13 cases).
3. Moderate cases: stridor and recession with various degrees of restlessness and reduced air entry on auscultation (27 cases).

In those who needed intubation to overcome their obstruction, the decision to intubate was made on entirely clinical grounds: those classified as severe, and those in the moderate group whose condition deteriorated on conservative management. This was usually indicated by a rising pulse and respiratory rate, and increasing recession and restlessness.

Conservative treatment consisted of moist oxygen, sedation, fluids, physiotherapy and antibiotics. Intubated patients were usually extubated on the 7th day. Most of the children in all groups had some degree of pneumonia and a few were in cardiac failure.

METHODS

Arterial blood was obtained by femoral artery puncture on admission and, where possible, daily thereafter. In those who were intubated, arterial blood was repeated 1 hour after intubation. All specimens were taken while the patient had been breathing air for at least 10 minutes.

The arterial carbon dioxide tension (PaCO₂) was measured by the interpolation method, and arterial oxygen tension (PaO₂) by an electrode, within 30 minutes of the blood being drawn.

RESULTS

The mean values of the blood gases and acid base of each clinical group were calculated on clinical assessment on admission (O/A), after intubation (A), on day 2 (D2), day 4 (D4), and on day 7 (D7). The moderate group was divided into those who subsequently needed intubation (15 cases), and those for whom conservative management was adequate (12 cases).

The normal values in our laboratory for children of a like age are pH 7.415 ± 0.043, PaCO₂ 37 ± 4 mmHg, base excess -0.4 ± 2, and PaO₂ 87 ± 5 mmHg.

**Fig. 1. Arterial pH of the clinical groups.**

On Assessment

The PaO₂ values of all 3 groups were significantly different from each other (P < 0.01), but that of the mild group was not different from normal.

The PaCO₂ of the mild group was significantly lower (P < 0.05) than normal, and also lower than the moderate group (P < 0.01). The difference between the moderate and severe groups was not significant.

The pH of the mild group was not significantly higher than normal, but was higher than that of the moderate...
TABLE I. DAILY BLOOD GASES

Means and standard deviations

<table>
<thead>
<tr>
<th>Clinical group</th>
<th>O/A*</th>
<th>A†</th>
<th>D2‡</th>
<th>D4‡</th>
<th>D7‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pH</td>
<td>PaCO₂</td>
<td>BE</td>
<td>PaO₂</td>
<td>pH</td>
</tr>
<tr>
<td>Severe</td>
<td>7,322</td>
<td>±0,102</td>
<td>31 ±12</td>
<td>-4,4 ±7</td>
<td>7,386</td>
</tr>
<tr>
<td>Moderate intubation</td>
<td>7,385</td>
<td>±0,079</td>
<td>33 ±10</td>
<td>-4,0 ±7</td>
<td>7,419</td>
</tr>
<tr>
<td>Moderate conservative</td>
<td>7,443</td>
<td>±0,071</td>
<td>32 ±5</td>
<td>+1 ±3</td>
<td>7,448</td>
</tr>
<tr>
<td>Mild</td>
<td>7,443</td>
<td>±0,050</td>
<td>32 ±3</td>
<td>+0,9 ±3</td>
<td>7,435</td>
</tr>
</tbody>
</table>

* O/A = on assessment.
† A = 1 hour after intubation.
‡ D2, D4, D7 = Day 2, Day 4, Day 7.

Progress of Disease

The most outstanding finding was that the patients all sought to make their blood alkaline, and that they did this by whatever means were at their disposal. If they could they seemed to prefer to do this by hyperventilation, but if this was not possible, they developed a metabolic alkalosis.

pH: By 24 hours the pH of the total material was significantly above normal (P < 0,01). This entailed a rise in three-quarters of the patients from the various admission values (Fig. 1). The pH remained high for 2 - 3 days and then fell to normal.

PaCO₂: Immediately following intubation in the severe and moderate intubated patients, the mean PaCO₂ fell significantly below normal (P < 0,05), and on D2 was still below normal (P < 0,01). Thereafter there was a slow rise towards normal. The level of the mild cases was significantly (P < 0,05) below normal from D1 to D3 and then rose. The moderate conservative group exhibited no change in mean values from D1 to D2, and thereafter a slow fall.

BE: The moderate conservative group showed a significant rise in BE (P < 0,05) over the first 24 hours, after which the level fell over the week of study. The other significant rise (P < 0,05) over the first day occurred in the severe group. Mean values of the mild and moderate intubated groups showed little variation over the 7 days.

PaO₂: In the severe patients intubation did not permit an immediate rise in PaO₂ but by D2 the rise was significant (P < 0,05). Intubation of moderate patients led to a fall in oxygen tension followed by a rise within 24 hours. In these 2 intubated groups the changes in PaO₂ correlated significantly (P < 0,001) with their BE changes in the first 24 hours (BE = 0,15, PaO₂ + 0,27, r = 0,62). The moderate conservative patients did not alter their mean PaO₂ from D1 to D2. In all patients the PaO₂ rose towards normal, except the mild cases whose level fell slightly over the week.

DISCUSSION

The patients in this study were graded on clinical grounds alone into 3 severity groups. The mean values of arterial oxygen tension and acid-base were calculated after the study was completed. In several cases these means were significantly different from each other, indicating that the features used in clinical assessment were helpful.
From the admission acid-base studies it is evident that the more severe the disease, the greater the acidemia, which was secondary to both metabolic and respiratory acidosis. On clinical grounds the 2 groups at either end of the severity spectrum could be easily recognized. With moderately ill patients there may be difficulty in selecting treatment on clinical grounds. Although serial blood examinations were helpful, the admission blood examination did not aid in the separation of this group into those who could be managed conservatively and those who subsequently needed intubation.

Laryngotracheobronchitis is a disease of upper airway obstruction. Yet the tendency of patients was not to underventilate. Only 26% of the total material, and 43% (12 of 28 cases) of those patients severe enough to require intubation were hyperventilating. In fact, there was apparently a preference to hyperventilate. This was illustrated by the striking fall in carbon dioxide tension immediately after the obstruction was bypassed by intubation. The hyperventilation continued for some days. Further illustration is evident in the mild group, who were hyperventilating on admission.

The reason for the hyperventilation was not immediately obvious. Fever, anxiety, hypoxia, or the presence of an endotracheal tube were not the cause, for they were not features of the mild cases whose arterial carbon dioxide tension was as low as that of the more severe cases. Acidemia and hyperventilation were not related because the mean base excess of these patients with a low (< 32 mmHg) and those with a normal carbon dioxide tension were not significantly different. Thus it seemed that these patients were not hyperventilating for any of the usual reasons.

In addition to hyperventilation, the pH of the intubated patients was raised when oxygenation allowed their haemoglobin buffer system to function. Evidence of this was the striking correlation of PaO and BE in the 24 hours following intubation.

Of all the patients, only 1 subgroup, the moderate conservative, did not hyperventilate. These were patients who struggled on with a considerable degree of upper airway obstruction, but who never came to intubation. This group raised their pH as far and as fast as did all other groups, but by means of a significant rise in their mean base excess during the first 24 hours. This manoeuvre was not related to carbon dioxide or oxygen tension, as neither changed over this period. In addition, the calculated mean oxygen content of this group did not alter significantly.

Explanation of the metabolic alkalosis was not evident. External loss of acid, as in vomiting, did not occur. These patients had a high intake of alkali, in the form of milk feeds, but so did the other groups. The derivation of the mean values for this group might have obscured multidirectional acid-base changes with a fortuitous mean in the alkali region. Therefore, the details of each case were re-examined. But the rise in base excess in the majority of cases could not be explained by conventional renal or extrarenal mechanisms.

Two mechanisms might have led to the metabolic alkalosis of these patients. Five-sixths of the body’s buffer capacity exists outside the intravascular space. These cellular buffers might have been mobilized, but the stimulus is as yet obscure.

The second possibility is the development of intracellular acidosis with extracellular alkalosis. Hydrogen ion usually enters cells if there is a shift of potassium out of cells. The common causes of potassium loss were not operative in these patients, e.g. diuretics, diarrhoea, vomiting. Excess of steroids lead to potassium loss in the urine, and steroids may have a specific alkalinizing effect unconnected with potassium loss. These children were not given steroids, but an increase of endogenous circulating steroid results from many stress levels. Levels of circulating steroids during pharyngitis and pneumonia have been shown to increase five-fold above normal. Whether this degree of endogenous steroid secretion could lead to alkalinization is not established, but deserves consideration.

In this study patients with laryngotracheobronchitis were found to manipulate their acid-base status by 3 different mechanisms. The majority hyperventilated. In some this could only be achieved with the assistance of intubation. The minority of patients who did not, or could not, hyperventilate, developed a metabolic alkalosis. Thirdly, the intubated patients utilized their haemoglobin buffer system. The common result of the acid-base changes was a rise in arterial pH. The pH attained, however, was not normal, but significantly above normal. This suggests that in treating group it might be necessary to correct the arterial pH to a figure higher than normal.

I should like to thank Dr R. Holloway, Physician-in-charge of the Respiratory Unit, King Edward VIII Hospital, for criticism and encouragement, and the Medical Superintendent, King Edward VIII Hospital, for facilities.

REFERENCES