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ORIGINAL ARTICLE

Acid-Base Balance and Blood-Gas Analysis in Bronchopneumonia in Infancy and Childhood

by

MARDJANIS SAID, I. BOEDIMAN, NASTITI N. RAHAJOE and NOENOENG RAHAJOE

(From the Department of Child Health, Medical School University of Indonesia, Dr. Cipto Mangunkusumo Hospital, Jakarta)

Abstract

Arterial Blood-Gas and pH changes were studied in sixty-three infants and children aged 6 years or less, with severe and uncomplicated bronchopneumonia, which was proven clinically and radiologically.

Fifty-five (87.3%) patients showed a ventilatory insufficiency (PaC02 below 35 mmHg). Only 3 (4.8%) cases were found with carbon retention as a sign of ventilatory failure (PaC02 above 45 mmHg). Twenty-eight (44.4%) cases had metabolic component. The suggested causes of metabolic acidosis in these cases were the inflammatory process in the lungs itself which caused hypoxia, and a low intake during their sickness prior to admission to the hospital.

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In 33 cases oxygen 3 litres per minute by nasal catheter had been administered at the time the blood sample was taken; 14 (42.4%) out of these 33 cases were still found to be with hypoxemia (PaO2 below 80 mmHg). But in the other 30 cases in whom oxygen was not given, all were with hypoxemia.

In these 30 cases we tried to correlate between the hypoxic sign and oxygen saturation (S.02), and only cyanosis was found to be the most precise clinical sign of hypoxia. Restlessness and impairment of conciousness were not reliable indices of hypoxia.

Our findings seems to support the suggestion that the clinical signs of respiratory distress were found to be of no value as an indication of the blood-gas levels in bronchopneumonia in infancy and childhood.

Introduction

Bronchopneumonia is one of the most common and severe respiratory disorders encountered in infants and children.

Like most of all acute pulmonary diseases, bronchopneumonia leads to inflammatory changes in the tracheobronchial tree and the alveoli; and this in turn leads to collapse of alveoli, pooling of secretions and uneven distribution of ventilation. The resulting intrapulmonary shunt causes arterial hypoxemia. The ventilatory system increases its work to compensate hypoxemia and in doing so causes a decrease in arterial PCO2. When oxygen is not available in tissues, metabolism continues through metabolic pathways known as "anaerobic pathways". The metabolic product of these pathways is lactic acid, which enters the venous blood and has a profound effect on the blood pH. The result is lactic acidosis.

Simpson and Flenley (1967) noted that among 1100 children under three years of age admitted to the Royal Hospital for Sick Children, Edinburgh, between 1960 and 1966 with acute lower-respiratory tract infection the mortality rate was 4.6%.

In the Department of Child Health, Medical School, University of Indonesia, Jakarta, 922 patients in the group of 0-14 years suffered from bronchopulmonary diseases in 1974, 812 patients in 1975 and 936 patients in 1976, and the mortality rates were 25%, 28% and 26% respectively (Rachmat Sadeli et al., 1978).

However, only minimal physiologic information exists concerning the severe bronchopneumonia in pediatric patients. Knowledge of the alteration of ventilation and acid-base balance encountered in this disorder is essential to design an effective therapeutic program besides causative treatment.

The present study was undertaken to determine the magnitude of arterial blood gas and acid-base derangements, and to correlate these findings with the clinical picture of this respiratory disorder.

Material and method

Sixty-three infants and children suffering from bronchopneumonia admitted to the Child Health Department, Dr. Cipto Mangunkusumo Hospital, Jakarta, between the period of January 1974 to December 1976, will be analysed to the acid-base balance and blood gas data. Only severe and uncomplicated cases admitted during the work hour were investigated.

The average age was 1.59 years with a range of 1 month to 6 years. Sex distribution was 37 males and 26 females.

Blood sample was obtained within thirty minutes of admission with or without oxygen administration (3 litres per minute of flow with nasal catheter); oxygen was given initially based on clinical grounds without blood-gas data. In all cases blood was taken in a hepa-

rinised syringe from the femoral artery. No sequelae other than minor hematomas resulted from these punctures.

In some cases subsequent blood and gas samples were taken after 24 hours of treatment. All blood samples were analysed in the Laboratory of Intensive Care Unit of the same hospital for pH and arterial carbon dioxide tension (PaCO2) by the method of Astrup et al. (1960), using Blood Micro Equipment Type BME 31 made by Radiometer A/S Corporation, Copenhagen, Denmark. The PaCO2 and base excess or deficit (negative base excess) were calculated using the Sigaard-Andersen nomogram after correction for hemoglobin saturation.

Besides that we also determined total bicarbonate (HCO3), Standard bicarbonate (S-bic), Buffer base (B.B.), arterial oxygen tension (PaO2) and oxygen saturation (S.O2).

The apparatus used were Acid-Base Cart ABC-1, Gas Mixing Apparatus Type GMAI/O, Blood Micro System Type BMS 3 and Acid-Base Analyser Type PHM 71.

The clinical assesment was done within fifteen minutes of admission to hospital. Any restlessness, cyanosis and impairment of consciousness were also noted. Chest X-ray, routine laboratory examination and tuberculin skin test were done in all cases. Conventional therapy with intravenous fluid drip (I.V.F.D.), humidified oxygen and antibiotics was always used. Assisted ventilation was used if necessary.

Based on blood gas data, cases with acidosis were corrected with sodium bicarbonate. In order to maintain an arterial pH between 7.35 and 7.45, intravenous sodium bicarbonate was administrated in a dose calculated according to the following formula : $NaHCO3\ (mEq) = 0.3 \times \text{body weight (kg)} \times \text{Base Deficit (mEq/L)}$. Half of the amount was given initially and half in 24 hours drip. This dose was intended to completely compensate the base deficit in the estimated extracellular space which was assumed to be 25 to 30% of the body weight.

Results and discussion

Fifty out of the 63 patients recovered completely, 13 (20.7%) patients died during the study, 8 patients died before 24 hours of hospitalization and 5 patients after more than 24 hours of hospitalization. No necropsy was done.

The distribution of ventilatory disturbances on admission are shown in table 1. Based on the categories of ventilatory disturbances (Shapiro, 1976), we noted that 55 patients (87.3%) showed a ventilatory insufficiency (PaCO2 below 35 mmHg) on admission.

Out of these 55 patients, 8 patients (12.7%) were with acute ventilatory insufficiency (accompanied by pH above 7.45), 26 patients (41.3%) with chronic ventilatory insufficiency (accompanied by pH between 7.35 - 7.45) and the

other 21 patients (33.3%) had ventilatory insufficiency combined with metabolic acidosis (accompanied by pH below 7.35).

It is in contrast to Reynolds' (1963) and Simpson and Flenley's (1967) observations that severe respiratory failure is not uncommon in such cases. Carbon retention as a sign of ventilatory failure (PaCO2 above 45 mmHg) was found only in 3 cases (4.8%). And all these cases were combined with metabolic acidosis (accompanied by pH below 7.35).

Five other cases had normal ventilatory status (PaCO2 between 35 - 45 mmHg) but 4 of them (6.3%) were combined with metabolic acidosis (accompanied by pH below 7.35).

TABLE 1 : *The distribution of ventilatory disturbances*

	$PaCO_2$	pH	(N)	%
acute ventil. insuff.	< 35	> 7.45	8	12.7
ventil. insuff. + metab. acidosis	< 35	< 7.35	21*	33.3
chr. ventil. insuff.	< 35	7.35 - 7.45	26	41.3
"n o r m a l"	35 - 45	7.35 - 7.45	1	1.6
normal ventil. status + metab. acidosis	35 - 45	< 7.35	4*	6.3
acute ventil. failure + metab. acidosis	> 45	< 7.35	3*	4.8
T o t a l			63	100.0

* 28 cases with metabolic acidosis

So in all of our series we found that 28 cases (44.4%) had metabolic component. Table 2 showed the range of pH, B.E. and bicarbonate of metabolic acidosis group in comparison with non-metabolic group patients. We found significant differences between the values of pH, B.E. and bicarbonate in the 2 groups.

TABLE 2 : *The range of pH, B.E. & Bicarbonate*

	Metabolic acidosis		P
	(+)	(—)	
(N)	28	35	
pH	7.26 ± 0.07	7.42 ± 0.18	< 0.001
B.E.	—12.7 ± 4.7	— 6.8 ± 3.28	< 0.001
Bic.	13.6 ± 4.41	15.8 ± 3.39	< 0.05

There were some factors that may cause metabolic acidosis in these patients. Huckabee (1958) stated that when oxygen is not available to tissues, anaerobic metabolism with production of lactic acid can produce a metabolic acidosis.

And in our series, all the 30 patients (100%) in whom oxygen was not yet administered at the time blood sample was taken on admission, were in hypoxemia (PaO2 below 80 mmHg); whereas 14 (42.4%) out of 33 patients on whom oxygen was given, 3 litres per minute of flow with nasal catheter, on admission before blood samples were obtained for analysis, failed to make PaO2 to normal levels (table 3).

TABLE 3 : *O_2 3L/min*

$PaCO_2$ (mmHg)		(N)
< 80	⩾ 80	
14 (42.4%)	19 (57.6%)	33

This finding was in contrast to Simpson and Flenley's report (1967) and we support the contention that hypoxia was a potent cause of a metabolic acidosis in these patients.

Most of our cases suffered from malnutrition. Forty-seven (74.6%) cases had less than 80% P. 50 of Stuart Chart (Harvard) percentile of body weight (table 4).

TABLE 4 : *Body weight on admission*

Body weight	(N)	%
< 80% P. 50*	47	74.60
80% P.50 - P.50	14	22.22
> P.50	2	3.18
T o t a l	63	100.00

* P.50 Stuart (Harvard) standard

All of our cases had a low intake during their sickness prior to admission to hospital. And the history of sickness prior to admission which anamnestically taken from their parents revealed the ranged from 1 - 14 days of duration with the averaged of 4.5 days.

These factors had also important roles in producing metabolic acidosis (starvation or keto-acidosis) in our patients. So it is suggestive that the causes of metabolic acidosis in our patients were hypoxia and starvation due to low intake during sickness.

It seems reasonable to assume that oxygen therapy has been effective as seen in arterial PO2 which were within normal range, and we therefore regarded a PO2 of 80 mmHg or more as meeting requirements. Nineteen (57.6%) cases out of 33 patients in whom oxygen was given on admission were with corrected hypoxemia (PaO2 above 80 mmHg). But unfortunately, because of lack of facilities, an inspired oxygen concentration (FiO2) was not done.

According to Simpson (1966) a 3 - 4 litres per minute oxygen flow can produce FiO2 of 40% in incubators. Reynolds (1963) suggested that FiO2 of 40% can raise the PaO2 above 100 mmHg in babies with bronchiolitis.

In the other hand Simpson and Flenley (1967) also reported that the administration of 40% failed to produce normal levels of PaO2 in 4 cases (19%). Therefore, we also assumed that 40% oxygen will always be sufficient for treating lower respiratory tract infection in infancy and childhood.

All patients of our series (100%) exhibited flare of alae nasi, lower chest retraction, tachypnea, tachycardia, fever and rales in both lungs and some of them showed cyanosis, restlessness and impaired consciousness from apathy to somnolence. So we tried to correlate between these hypoxic signs : cyanosis, restlessness and impairment of consciousness and S.O2 (table 5).

TABLE 5 : *The correlation between hypoxic signs and $S.O_2$*

	Cyanosis		Restlessness		Impairment of consciousness	
	(+)	(—)	(+)	(—)	(+)	(—)
$S.O_2$ (%)	78.3 ± 5.76	92.6 ± 3.15	86.1 ± 8.28	83.3 ± 8.72	85.4 ± 9.13	84.5 ± 8.61
(N)	16	14	18	12	17	13
P	<0.001		> 0.05		> 0.05	

In this study we found that there was a significant correlation between cyanosis and S.O2 ($P < 0.001$). Because in these 30 cases with cyanosis the arterial S.O2 was always below 90% and most often below 85%.

But we also found that there were no significant corelation between restlessness and S.O2 and between impairment of consciousness and S.O2 ($P > 0.05$).

So our findings lend no support to the idea that restlessness and impairment of consciousness are valid clinical signs of hypoxia of such cases. But we support the suggestion of Simpson and Flenley (1967) that of the 3 clinical signs of hypoxia that we have discussed, only cyanosis seems to be of value as a guide to the S.O2.

In the other hand Morrison (1955) suggested that restlessness was a valuable sign of hypoxia in such cases. Morrison determined the oxygen saturation on "arterialised" capillary blood on thirty-one occasions in 18 children and found that restlessness was much more often present when the saturation was below 80%.

It is possible that, the capillary blood was not an adequate substitute for arterial blood, at least in regard to its S.O2.

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