Acta Paediatrica Academiae Scientiarum Hungaricae Vol. 7 (3), pp. 293—300 (1966)

Blood Gases and Blood pH in Spastic Bronchitis of Infants

By

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(Received April 18, 1966)

This study had the purpose to observe changes in the blood gases, in the value of blood pH and in the acid-base equilibrium of infants caused by spastic bronchitis and by oxygen therapy.

It is fairly difficult to distinguish between the different grave diseases of the lower respiratory tract of infants, e.g. between bronchiolitis and bronchopneumonia. This applies also to spastic bronchitis, an infectious disease frequently accompanied by grave dyspnoea [2].

Although infantile bronchitis may be of various aetiology, its clinical manifestations are fairly uniform: the cough is insignificant or absent; inspiratory and expiratory dyspnoea is usually serious but responds readily to treatment with spasmolytic drugs and cortisone. The course of the disease and the prompt therapeutic result offer good clues for *a-posteriori* diagnosis. Table I shows the principal clinical symptoms encountered in the material of this study.

Material and Method

The material consisted of 15 babies aged from 1 to 16 months. Serial numbers refer to the same patients in all Tables. The highest rectal temperature was 38.6°C, but only three infants had fever exceeding 38°C. Respiration rate was between 40 and 80/min., mean, 56. The average pulse rate amounted to 145/min. All the patients presented both expiratory and inspiratory serious dyspnoea. All but four were cyanotic. The sensorium was confused in the gravest cases, while the others displayed symptoms of grave hypoxia such as restlessness and alarm. (Restlessness is considered a more reliable sign of hypoxia than cyanosis [5]). Six patients had unilateral, and two bilateral, maxillary sinusitis; puncture yielded copious purulent discharge in all these cases. X-ray revealed deeply situated diaphragms, and rich perihilar markings in otherwise clear lung fields. In addition, moderate atelectasis was found in the lower lung fields of four patients. Auscultation yielded poor findings; apart from prolonged expiration, moderate bubbling rales were heard in some cases. Treatment with cortisone and antibiotics usually relieved the dyspnoea in 24 hrs, and ensured full recovery in five to six days. There was one exception: a baby of 7 weeks who had been moribund at admission died two days later. (Table 1.)

Oxygen was administered by means of a glass funnel placed above the face of the babies so as to allow the taking of blood samples from the temporal arteries. The percentage composition of the oxygen was, therefore, variable. Blood was usually drawn at admission (i.e. prior to treat-

TABLE Ia

Serial No.	Age (months)	Weight (kg)	Temper. (C°)	Frequency of respir.	Pulse rate	Dyspnoea 1
1	2	3.1	36.9	60	160	++
2	3.5	1.9	37.8	52	150	+
3	3	5.4	37.1	40	132	++
4	3.5	4.1	38.6	72	180	+
5	12	9.4	38.5	76	112	+
6	4	5.4	36.8	40	120	++
7	8	6.6	38.1	60	160	+
8	6	7.2	37.2	48	160	+
9	4	2.4	36.5	60	120	+
10	1.8	2.2	36	56	160	++
11	1.5	3.4	36.6	54	120	++
12	3	3.6	37.0	60	160	+
13	1	3.3	36.5	68	160	+
14	16	8.1	37.6	30	150	++
15	3	3.5	36.6	60	144	++
Average	5 ±4.1			56 ±12.2	145 ±20.3	

1. + pronounced
++ grave

ment) and then in the fifth minute of O_2 administration. O_2 tension, arterial pH and the other parameters were determined by means of Astrup's apparatus [1], while SCHOLANDER's micromethod [6] was used for the volumetric determination of blood gases. CO_2 tension was computed by NUN's interpolation technique with the SIGGAARD-ANDERSEN nomogram (3,8).

RESULTS

Values for blood gases, blood pH and other parameters found before treatment are assembled in Table II.

(i) Oxygen tension (pO_2) was decreased in all patients, and even conspicuously low in certain cases. O_2 saturation of the arterial blood was correspondingly low in most cases. O_2 saturation below 70 per cent is known to cause grave hypoxia and is frequently fatal in adults. Mean arterial O_2 saturation was 66 per cent in the present material; despite the hypoxia there was but a single fatal case, and it was not the baby with the lowest oxygen saturation.

(ii) Carbon-dioxide tension (pCO_2). Low, normal and high pCO_2 values were equally found. Values between 34 and 45 mm Hg were considered normal [7]. Sequence in Table II is arranged according to pCO_2 values.

(a) Three babies had low pCO_2 values. The level of standard bicar-

TABLE Ib

Cyanosis	Behaviour	Maxill. sinusitis	Chest radiogr. 2	Notes
+	restless	−	+ atelectasis	recovered
+	restless	−	+	recovered
+	restless	+	+ atelectasis	recovered
+	restless	+	+	recovered
−	restless	+	+ atelectasis	recovered
+	restless	+	+	recovered
+	disturbed	−	+ atelectasis	recovered
−	disturbed	+	+	recovered
−	restless	++	+	recovered
+	disturbed	+	+	died
−	restless	−	+	recovered
+	restless	−	+	recovered
+	restless	−	+	recovered
+	restless	++	+	recovered
+	restless	−	+	recovered
				+ congenital defect

2. + radiogram showing the characteristic features of spastic bronchitis

bonate was low and the base excess pronounced, indicative of metabolic acidosis presumably caused by hypoxia. Standard bicarbonate values between 21.3 and 24.8 mEq/litre and a base excess between −2.3 mEq/l and + 2.3 mEq/l were considered normal. Metabolic acidosis was present in all patients. This explains why the low pCO_2 was not accompanied by respiratory alkalosis, while the blood pH reached the uppermost limit of normal in one case.

(b) *A normal pCO_2* was found in five babies. Low blood pH, low standard bicarbonate and high base excess, i.e. the signs of metabolic acidosis were present in these cases also. A normal pCO_2 associated with acidosis of this degree means that respiration is unable to adapt itself to the existing acidosis.

(c) *A high pCO_2* occurred in half of the patients, and it was this group which exhibited the highest mean value and the lowest individual value of blood pH (7.245 and 7.09, respectively). The fact that — with a single exception — standard bicarbonate and base excess were low admits of the conclusion that metabolic acidosis associated with and intensified by respiratory acidosis in this group.

As regards changes caused by a five-minute O_2 inhalation, the material could be divided in three groups.

TABLE II

Serial No.	pCO_2 mm Hg	pO_2 mm Hg	Art. O_2 satur. %	Haemogl. g per 100 ml	pH	Standard bicarbonate mEq/litre	Base excess mEq/litre	Buffer base mEq/litre
5	25.5	38	51	7.9	7.45	16.6	− 8	30.2
8	26	74	44.5	10.1	7.342	16.5	−10	30.2
a) 11	30.5	78	66	6.6	7.27	15.2	−12	37
Average	27.3	63	53.8	8.2	7.354	16.1	−10	35.1
	±2.7	±22	±11.1	±1.7	±0.09	±0.8	±2.0	±4.3
4	42	46	61	8.7	7.27	18.2	− 7.8	42
7	41.5	50	65	9.4	7.295	19.2	− 6.5	42.5
b) 13	43	69	72	12.7	7.21	17.2	− 6.5	27.5
14	44.5	58.5	77.5	12.3	7.225	17	− 9	34.2
15	42.2	78	89	12.0	7.39	17.5	− 7.5	32
Average	42.2	60.3	72.9	11.0	7.278	17.8	− 7.4	35.6
	±1.27	±13.3	±11.0	±1.8	±0.07	±0.56	±1.04	±6.5
1	57	42	59.5	13.4	7.21	20	− 4.3	30
2	47.5	50	56	13.6	7.33	22	− 2.9	51.5
c) 3	45.5	68	55	13	7.24	17.8	− 8.2	40
6	56	59	75	10.8	7.305	25.2	+ 1.8	55
9	62.5	43	63	11.1	7.282	22.5	− 2.0	51
10	64	78	61	9.3	7.09	14.6	−13.8	36
12	48	81	92	8.2	7.26	19	− 7	44
Average	54.3	60	66	11.3	7.245	20.1	− 5.2	44
	±7.4	±16	±13.2	±2.1	±0.08	±3.4	± 5.0	±9.1

(a) Table III presents data of ten babies in whom an increased arterial pO_2 was accompanied by a decreased pCO_2. Both the increase and the decrease were significant statistically ($p < 0.1$ per cent). Arterial pH rose from 7.255 to 7.28. This readjustment of the blood gases means the near accomplishment of the desired therapeutical result, i.e. the relief of hypoxia by increasing pO_2 and O_2 saturation, the elimination of respiratory acidosis and the compensation of metabolic acidosis by a reduction of the high pCO_2.

(b) Table IV present values for four cases in which the increase in arterial pO_2 was accompanied by an increase in pCO_2. The elevation of pO_2 was significant statistically ($p < 0.1$ per cent), and it was considerable also in respect of the pCO_2 ($p < 5.0$ per) cent. Arterial pH decreased. The initial pCO_2 value was subnormal in two cases and did not exceed the normal level even after

TABLE III

Increasing pO_2, decreasing pCO_2

Before O_2 inhalation After

Serial No.	pO_2 mm Hg	pCO_2 mm Hg	Art. O_2 satur. %	pH	pO_2 mm Hg	pCO_2 mm Hg	Art. O_2 satur. %	pH
1	42	57	59.5	7.21	50	56	67	7.22
2	50	47.5	56	7.33	66	40	61	7.36
4	46	42	61	7.27	77	25.3	80	7.35
7	50	41.5	65	7.295	84	37	86	7.325
10	78	64	61	7.09	186	53	75.5	7.13
11	78	30.5	66	7.27	204	29	100	7.28
12	81	48	92	7.26	206	41.5	94.5	7.29
13	69	43	72	7.21	140	40.5	82	7.245
14	58.5	44.5	77.5	7.225	137	37	90.5	7.275
15	78	42.2	89	7.39	80	37.5	94	7.33
Average	63	46	70	7.255	123	39,6	83	7.28
	±15.3	±9.0	±12.6	±0.08	±59.4	±9.3	±12.6	±0.06

$p < 0{,}1\% < 0{,}1\%$

TABLE IV

Increasing pO_2, increasing pCO_2

Before O_2 inhalation After

Serial No.	pO_2 mm Hg	pCO_2 mm Hg	Art. O_2 satur. %	pH	pO_2 mm Hg	pCO_2 mm Hg	Art. O_2 satur. %	pH
5	38	25.5	51	7.45	45	31	60	7.36
6	59	56	75	7.305	103	62	85.5	7.295
8	74	26	44.5	7.342	152	32	75	7.290
9	43	62.5	63	7.282	124	80	94	7.238
Average	53.5	42.5	58	7.344	106	51	79	7.295
	±16.3	±19.5	±13.4	±0.07	±45.3	±24	±14.6	±0.05

$p < 0{,}1\% < 5\%$

TABLE V

Decreasing pO_2, decreasing pCO_2 after O_2 inhalation

Serial No.	Before: pO_2 mm Hg	Before: pCO_2 mm Hg	Before: Art. O_2 satur. %	Before: pH	After: pO_2 mm Hg	After: pCO_2 mm Hg	After: Art. O_2 satur. %	After: pH
3	68	45.5	55	7.24	60	38.8	44.5	7.28

its rise. This value was *a limine* above normal in another two cases, O_2 inhalation in this group elevated the CO_2 level instead of reducing it, evidently because breathing — which, so far, had been able to perform its function — deteriorated still further, thus upsetting the mechanism of CO_2 exchange. Arterial pCO_2 may reach dangerous heights in such cases.

(c) Table V presents a case in which both arterial pO_2 and pCO_2 decreased. This baby, becoming restless on inhaling O_2, did not receive as much O_2 as would have sufficed to raise the arterial pO_2, while increased breathing intensified by excitement, eliminated an additional volume of CO_2 from the lungs.

In none of the cases was the decrease in pO_2 accompanied by increase in pCO_2. A phenomenon of this kind would not have been compatible with spastic bronchitis, since such changes in the blood gases point to a complete obstruction of the respiratory tract.

Discussion

We have seen that the effort of the organism to compensate metabolic acidosis caused by hypoxia resulted in a low arterial pCO_2 in not more than 20 per cent of the cases, while it failed to reduce pCO_2 in the majority. Inhalation of O_2 resulted in an elevation of pCO_2 in four cases, i.e. in nearly 25 per cent, a phenomenon that might cause some apprehension concerning the O_2 treatment of infantile bronchitis. Reynold [5] does not acknowledge the importance of this phenomenon, and suggests that a rise if pCO_2 need not interfere with O_2 administration.

It is known that the peripheral chemoreceptors for O_2 and CO_2 play an important part in the stimulation of the respiratory centre, especially in premature babies and infants [4, 9]. O_2 and CO_2 are presumably important factors in the maintenance of forced breathing in cases of spastic bronchitis; such breathing is of vital importance because it ensures that gas exchange without which no life is possible. It may happen that the improvement of hypoxia on O_2 inhalation deprives the organism of that important respiratory stimulus. Since, however, airway obstruction persists, the increase in pO_2 will be associated with an increase of pCO_2 because of the hypoventilation. The increased pCO_2 promotes breathing for some

time, but it will act in the opposite direction and paralyse above a certain limit the respiratory centre. It is, therefore, possible that in spastic bronchitis the inhalation of O_2 relieves the hypoxia, cyanosis and restlessness, while increasing accumulation of CO_2 causes sudden and fatal respiratory paralysis. This does not mean that O_2 administration must be dispensed with, the less so as hypoxia, too, may be fatal. If only follows that, when O_2 treatment is applied in diseases associated with airway obstruction, changes in arterial pCO_2 should carefully be followed.

Summary

Changes in the blood gases and in the acid-base balance, caused by a five-minute inhalation of O_2, have been studied in 15 infants suffering from spastic bronchitis. Grave hypoxia with low pO_2 and low arterial saturation was observed in nearly all babies before the inhalation of O_2. Low standard bicarbonate and high base excess values, encountered in nearly all cases, pointed to metabolic acidosis, presumably a consequence of hypoxia. The pCO_2 was below normal in about 20 per cent of the cases and normal or above normal in the rest, a sign showing that metabolic acidosis was accompanied by respiratory acidosis. In cases with a low pCO_2 the pH of arterial blood was around the lowest normal value since the patients were able to compensate metabolic acidosis by respiration. Disorders of ventilation impeded this compensation in the rest of the patient and their blood pH became correspondingly low.

O_2 inhalation yielded satisfactory results in 10 cases, as arterial pO_2 increased and pCO_2 decreased. The rise of pO_2 was associated with a rise of pCO_2 in four cases, presumably because stimulation of the respiratory centre and consequent ventilation, maintained during hypoxia via the peripheral chemoreceptors, were diminished by O_2 treatment, so that the constriction of the respiratory tract promoted the accumulation of CO_2. Continuous recording of the arterial CO_2 level in patients subjected to O_2 treatment is, therefore, imperative.

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