THE MANAGEMENT OF METABOLIC ACIDOSIS IN THE PAEDIATRIC AGE-GROUP

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Metabolic acidosis is the most frequent and important disturbance of acid-base balance in the paediatric age-group.[1] The various conditions associated with metabolic acidosis have been listed in an earlier article.[1] The aim of this paper is to discuss the principles and management of treatment in metabolic acidosis, and examples are given to illustrate the different aspects.

The management of acid-base disturbances should, in the first instance, be directed at the underlying illness and not only the acid-base findings at any given time. The management should therefore consist of:

(*i*) therapy dealing specifically with the primary cause of the illness;

(*ii*) measures which would promote a return to, or an improvement of, the usual homeostatic mechanisms for acid-base regulation; and

(*iii*) the administration of an acid or base to correct metabolic disturbances.

Specific Therapy

Standard textbooks can be consulted on specific therapy to deal with the primary cause of the illness, which will not be discussed in this article.

Measures to Promote Normal Homeostatic Mechanisms

It is unnecessary also to discuss at length measures which would promote a return to or an improvement of the normal homeostatic mechanisms for acid-base regulation, as they are covered under the management of the various illnesses in the textbooks. The importance of correcting dehydration and the establishment of normal renal function must, however, be stressed. Dehydration and associated impaired renal function is, for practical purposes, always present in sick infants and neonates who show evidence of acid-base disturbance. It is also more common in older children, especially those presenting with status asthmaticus, than is often suspected. It is met with both in situations associated with metabolic and with respiratory disturbances.

The importance of correcting dehydration and the improvement of the normal renal homeostatic mechanisms in the correction of an acid-base disturbance is well illustrated by the following case:

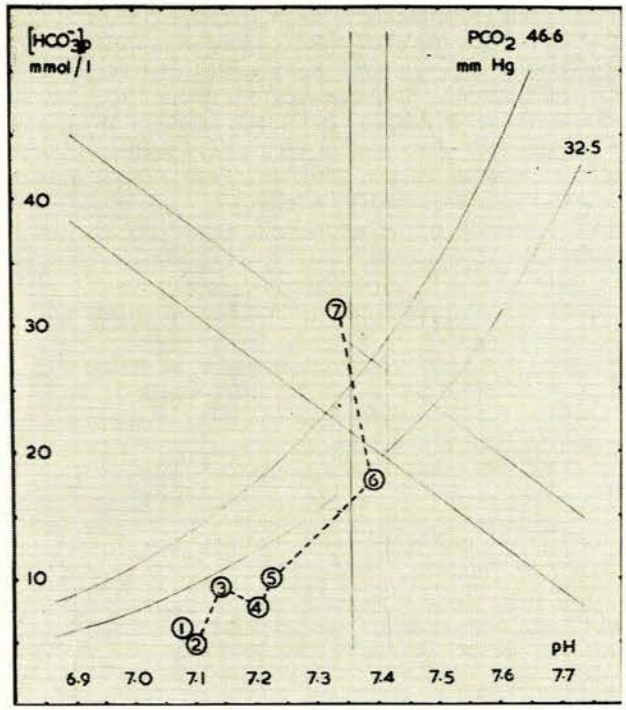
Infant G (Fig. 1), aged 9 weeks, suffered from gastroenteritis associated with severe dehydration and a profound metabolic acidosis. Following intravenous fluid and electrolyte therapy, the improvement in renal function and other homeostatic mechanisms resulted in a return to normal of the acid-base status of the infant without the administration of alkalizing agents. The changes in the acid-base status following rehydration over a period of 24 hours should be noted (Table I).

Administration of Acid or Base

The administration of an acid or a base is mainly indicated:

(*a*) as an emergency measure when the acid-base disturbance in itself is life-threatening and its rapid correction is a matter of urgency; or

(*b*) as a supportive measure when, because of the effects of the primary illness, the homeostatic mechanisms are inadequate and either temporarily or permanently deranged.



No.	0	1	2	3	4	5	6	7
Wt. (kg.)	3·41							4·12
Hours	0	½	1½	2½	3½	4½	10½	24
pH	7·05	7·05	7·09	7·12	7·16	7·16	7·38	7·32
PCO_2	16·2	16·2	20·2	24·2	19·5	21·8	23·3	53·0
BE	−25·0	−25·0	−23·0	−21·0	−21·0	−20·2	−9·4	−0·2

Fig. 1. See text.

TABLE I. CHANGES IN ACID-BASE STATUS FOLLOWING REHYDRATION

	pH	*PCO_2*	*BE*	*BB*	*Std. HCO_3*	*Act. HCO_3*
Arterial blood 0 hours	7·05	16·2	−25·0	21·4	7·8	6·8
Arterialized blood 24 hours	7·32	53·0	−0·2	48	23·8	26·8

Various workers have calculated the amount of acid or base that should be given to correct a metabolic disturbance of acid-base balance. The most widely used formula is that of Mellengard and Astrup[2] who found the BE in the extracellular space to be 0·3 of the body-weight in kg. The amount in mEq. of base or acid required to correct

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any disturbance of the acid-base status in the extracellular space is, therefore, calculated as follows:

Number of mEq. base or acid = BE in mEq./l. × 0·3 × body-weight in kg.

This amount may be given rapidly and the acid-base status should be checked ½ - 1 hour later. Further corrections can then be made according to the BE.

In dogs, the BE in the whole organism has been calculated at between 0·4 - 0·5 of the body-weight.[3] This formula should never be used for rapid administration as the acid or base takes much longer to equilibrate with the intracellular phase.

Children, and especially newborn infants, differ from adults in their body composition and some modifications to the above formula have been employed. Hutchison *et al.*[4] assumed that the extracellular space in newborn infants with RDS was 35% of the body-weight and used 0·35 in their calculations. According to Stahlman,[5] 60% of the newborn infant's body is water which needs to be buffered. Of the calculated amount (in this case of sodium bicarbonate) one-quarter to one-third was given rapidly and the remainder in a drip over 3 - 4 hours.

The following formulae for calculating the amount of bicarbonate to be administered have been employed in our unit over the last 2 years with good results:

Newborns and prematures: Number of mEq. acid or base = 0·4 - 0·6 × BE × kg. body-weight

Infants and older children: Number of mEq. acid or base = 0·4 - 0·6 × BE × kg. body-weight

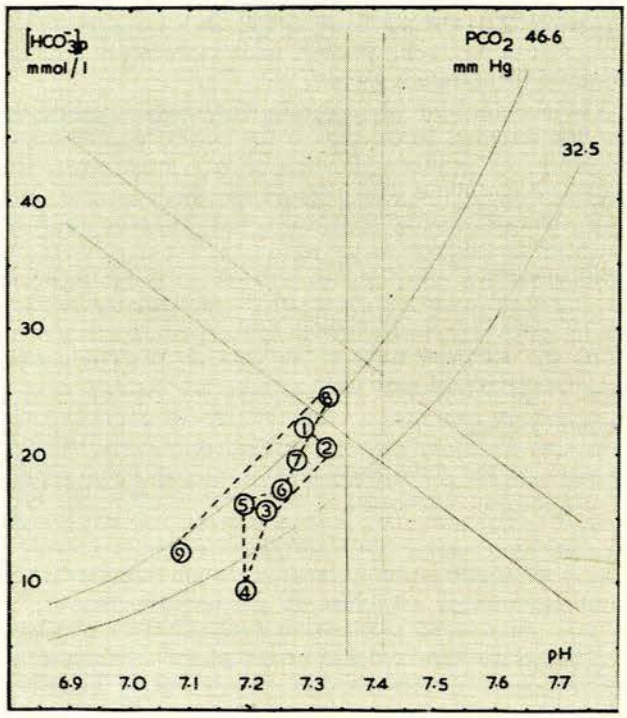
In most cases, one-quarter to one-third of the calculated amount is given rapidly. The remainder, however, is given at a slower rate than that suggested by other workers.

No untoward side-effects were encountered in spite of the sometimes large amounts of sodium bicarbonate administered to some of the premature infants. Baby S, aged 26 days, with a weight of 1·24 kg. (Fig. 2), received 32 mEq. of sodium bicarbonate over a 28-hour period, which is equivalent to giving a 70 kg. adult ± 1,800 mEq. or 3,600 ml. of 4% sodium bicarbonate over a similar period. Baby S developed severe respiratory distress soon after birth, but responded to oxygen therapy and intravenous tris (hydroxymethyl) aminomethane. The infant failed to thrive and at the age of 20 days was noticed to have an increased respiratory rate. Clinical, radiological, urinary and other special investigations failed to establish the cause of the failure to thrive and the increased respiratory rate. Acid-base studies from day 24 to day 31 showed the progressive development of a metabolic acidosis, which became partly compensated on day 31. The rapid correction of this metabolic acidosis was achieved over a period of 28 hours (Fig. 2) by the administration of 32·6 mEq. of sodium bicarbonate. The total calculated amount of sodium bicarbonate from the various base excess values over this period amounted to 32 mEq.

Following the cessation of bicarbonate therapy the infant progressively became more acidotic. A similar response was again achieved with bicarbonate therapy. Extensive investigations were carried out to elucidate the aetiology of the acidosis, including electrolyte, serum lactate and pyruvate estimations. Urinary pH electrolyte and chromatographic studies were undertaken without a conclusive diagnosis being achieved. The infant died suddenly at the age of 7 weeks. Postmortem studies revealed no abnormalities to account for the acid-base derangements.

A. ADMINISTRATION OF A BASE AS AN EMERGENCY PROCEDURE

It has already been stressed that, provided the kidney and the liver are normal, severe acid-base disturbances in cases of gastroenteritis can be corrected by applying the proven principles of fluid therapy (Fig. 1). In premature and/or malnourished infants with severe gastroenteritis, the combined effects of the preferential loss of bicarbonate in the stools and the acid-producing effects of starvation, infection, cellular destruction, renal and liver impairment, result in a profound acid-base derangement which may come on rapidly and constitute a threat to life.



No.	1	2	3	4	5	6	7	8	9
Age (days)	24	26	27	31					37
Hours				0	5	10	22	28	143
pH	7·30	7·31	7·21	7·23	7·20	7·25	7·27	7·34	7·09
PCO_2	44·4	39·0	40·0	22·2	43·4	40·4	41·4	46·0	38·0
BE	−4·1	−6·4	−11·7	−17·2	−10·0	−8·6	−7·2	−1·0	−16·4
Std. bicarb.	20·7	18·8	15·5	11·9	16·0	17·2	18·3	23·0	11·5
Bicarb. therapy					14·3		9·5	8·6	

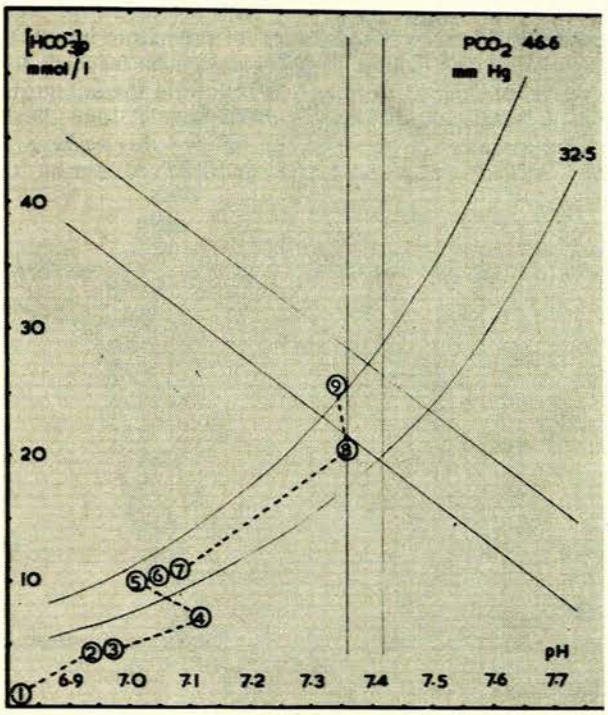
Fig. 2. See text.

Baby M.S., aged 6 weeks and weighing 1·94 kg., was born prematurely and had been discharged in a healthy state, (weighing 2·28 kg.) from a maternity hospital 5 days before she was first seen. She presented with diarrhoea which responded initially to treatment with intravenous fluids and antibiotics. Seven days later, however, she again developed severe diarrhoea and vomiting but only attended hospital 2 days later. Examination revealed a severely dehydrated, acidotic, semi-conscious and malnourished infant.

Investigations of blood obtained from the temporal artery were as follows: Hb 7·7 G/100 ml., pH 6·81, PCO_2 19·5 mm.Hg, BE > −30 (± 35), standard bicarbonate 4·5 mEq./l., serum sodium 152 mEq./l., chloride 127 mEq./l. and potassium 8·4 mEq./l.

She was given 20 mEq. of sodium bicarbonate intravenously over 30 minutes and intravenous therapy commenced with half Darrow's 2·5% dextrose solution with added sodium bicarbonate. Correction of the metabolic acidosis took place slowly and became normal (Fig. 3) after the administration of a total of 65 mEq. of sodium bicarbonate over 33 hours. At this time the infant was fully hydrated and weighed 2·15 kg. Investigation of the arterial blood revealed the following:

pH 7·35, PCO_2 = 47 mm.Hg, BE +0·5 mEq./l., buffer base 48·8, standard bicarbonate 28·7 mEq./l., actual bicarbonate 25·0 mEq./l., serum sodium 144·0 mEq./l., chloride 110·4 mEq./l. and potassium 7·0 mEq./l.



No.	1	2	3	4	5	6	7	8	9
Wt. (kg.)	1·94								2·15
Hours	0	½	1½	2½	3½	4½	10¾	21½	34
pH	6·81	6·95	6·95	7·13	7·03	7·07	7·05	7·36	7·35
PCO_2	19·5	17·5	17·5	16·8	36·0	30·0	32·0	35·0	47·0
BE	>−30	−26·0	−26·0	−22·1	−20·4	−20·4	−20·5	−5·0	+0·5

Fig. 3. See text.

She was fully conscious and sucked well. Following additional therapy she showed further improvement and was discharged apparently well.

B. ADMINISTRATION OF A BASE AS SUPPORTIVE THERAPY

The administration of a base such as sodium bicarbonate is often indicated in the management of 2 relatively common conditions affecting the premature infant, i.e. idiopathic respiratory distress syndrome (IRDS) and late acidosis of prematurity.

1. *Idiopathic Respiratory Distress Syndrome (IRDS)*

The impairment of ventilation and gas exchange in infants suffering from moderate to severe clinical hyaline membrane disease results in carbon dioxide retention and hypoxia. The latter results in cardiovascular disturbances, renal disturbances, anaerobic metabolism of tissues and in the insufficient metabolism with resulting accumulation of organic acids such as lactic acid, giving rise to a metabolic acidosis. The mixed acidosis provokes secondary electrolyte disturbances of hyperkalaemia, hyperphosphataemia and nitrogen retention. The work of breathing is increased to about 4 times the normal and the rise in expenditure of energy results in exhaustion of the glycogen stores.[6]

Corrective therapy must, therefore, be directed against:

(*a*) The impairment of ventilation, gas exchange and respiratory acidosis.

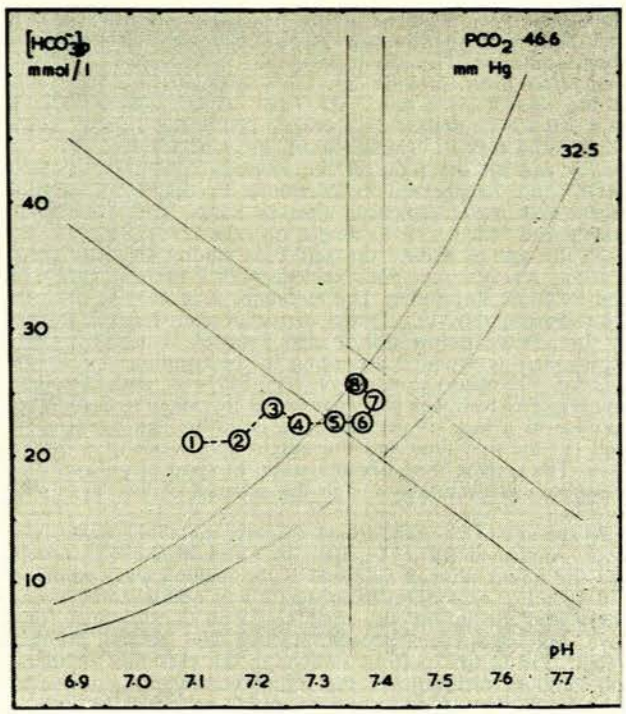
(*b*) Hypoxia.

(*c*) The metabolic acidosis.

Therapy is directed in the first instance against the hypoxia and the metabolic acidosis. As the latter is, in fact, secondary to a large extent to the hypoxia, atmospheric oxygen in concentrations of up to 100% is administered. An attempt is also made to supply readily available stores for energy production.

The intravascular administration of sodium bicarbonate is employed for the correction of the established metabolic acidosis or to neutralize any non-volatile acids still produced as a result of active disease.[4,5,7] Tham is also employed for the latter, but has a greater effect in the control of the respiratory acidosis.

The application of some of these principles in the management of clinical hyaline membrane disease is shown in baby W (Fig. 4), who was one of twins delivered by caesarean section because of severe antepartum haemorrhage. She weighed 1·89 kg. and showed evidence of severe respiratory distress soon after birth. This was manifested at the age of 4 hours by a rapid respiratory rate of 72, grunting, recession, oedema, sternal bulge, poor breath sounds and crepitations on auscultation of the lungs. Cyanosis could only be abolished by the administration of 100% oxygen. Radiological studies revealed a granular-reticular pattern and air bronchogram of both lungs. It was necessary to administer 100% oxygen to control the arterial oxygen tension within the range of 60 - 100 mm.Hg.



No.	1	2	3	4	5	6	7	8
Hours	4	11¼	18½	26½	38	44	61½	84½
pH	7·10	7·18	7·23	7·27	7·31	7·36	7·39	7·38
PCO_2	69·0	57·7	56·0	48·5	45·5	41·0	39·5	42·0
BE	−8·5	−7·2	−4·4	−4·8	−3·6	−2·2	−1·1	−0·4
Bicarb. therapy mEq		2·0	5·5	3·3	2·2	1·2	0·9	
PO_2	145	69	99	60	89			

Fig. 4. See text.

The calculated base deficit at the age of 4 hours was 9·6 mEq. Sodium bicarbonate in 10% dextrose solution was infused

intravenously at a slow rate and the progressive improvement and the return of the acid-base status to normal after a total administration of 14·2 mEq. of sodium bicarbonate over 40 hours is shown in Fig. 4. The infant eventually made a complete recovery.

2. *Late Metabolic Acidosis of Prematurity*

Kildeberg[8] demonstrated in 11 (8·6%) of 128 premature infants during the 2nd - 4th week of life, a metabolic acidosis characterized by:

(*a*) A clinical presentation of slow weight gain and poor feeding in apparently healthy premature infants fed with cow's milk.

(*b*) Laboratory evidence of a metabolic acidosis of gradual onset during this period with a tendency to spontaneous remission within weeks.

(*c*) A return to a normal acid-base status following a temporary withdrawal of the milk diet or by the administration of a few doses of bicarbonate.

(*d*) Improved feeding and gain in weight following the return to normal acid-base status.

Baby H.A., a female infant with a gestational age of 41 weeks according to the mother's dates, and weighing 1·95 kg., was delivered by elective caesarean section because of a previous history of ruptured uterus. The Apgar rating at delivery was 1/10 and spontaneous respiration was only established after intubation. The infant developed mild respiratory distress soon after birth, but did not show any acid-base disturbance at the age of 7½ hours: pH 7·283, PCO_2 46·8 mm.Hg, BE −4·8, BB 42·6. standard bicarbonate 20·2 mEq./l., actual bicarbonate 22·6 mEq./l. The X-ray of the chest revealed no abnormality and all the signs of the respiratory distress syndrome (RDS) had disappeared by 24 hours. Feeding with expressed breast milk was commenced after 24 hours. The infant sucked poorly and failed to gain weight on tube feeding.

At the age of 8 days the acid-base studies showed the presence of a severe metabolic acidosis with a pH of 7·14, BE −15 and PCO_2 of 30 mm.Hg. The metabolic acidosis was confirmed the following day (Fig. 5) and corrected over the next 24 hours by the administration in 8 divided doses of 22 mEq. of sodium bicarbonate (calculated according to the formula: 0·6 x −18 x 1·82 kg.=22 mEq.) to a pH of 7·35, BE + 1, and PCO_2 of 49 mm.Hg. The feed was also changed at this stage to S26 (Wyeth) because of a lack of expressed breast milk. The infant sucked well for the first time and the weight gain was sustained for 4 days. The weight then became static in spite of an apparently adequate caloric intake and in the absence of any signs of infection.

At the age of 22 and 23 days a severe metabolic acidosis was again confirmed (pH 7·19, BE −14·3 and PCO_2=33·5 mm.Hg) and the infant refused all feeds. Tube feeding was commenced and 18 mEq. of sodium bicarbonate was administered over 24 hours and the infant was maintained on 2 mEq. *b.d.* for 14 days. She again sucked well, thrived and gained 26 oz. in weight during this period. Following the cessation of therapy with sodium bicarbonate, the infant continued to thrive and gained a further 16 oz. in weight over a period of 13 days.

According to Kildeberg,[8] this late acidosis of prematurity is probably caused by a temporary disproportion between the renal capacity for hydrogen ion excretion and the daily load of non-volatile acid. Buchanan and Komrower[9] considered that the renal excretion of H^+ was normal in children if the urinary pH fell below 5·0 after the administration of NH_4Cl as a single dose of 0·1 G/kg. body-weight. Elkington *et al.*[10] found in adults that measurement of urinary pH was not enough to differentiate latent cases of renal acidosis and suggested that the qualitative response to an NH_4Cl load expressed as the H^+ clearance index should be employed to demonstrate latent acidosis. Peonides *et al.*[11] employed this method in infants over the age of 3 months. In the present case, at the age of 43 days following the administration in divided doses of 0·75 G of NH_4Cl per day for 3 days, the urinary pH was 5·3 and the H^+ clearance index 0·18. Although these values in older infants would probably be indicative of renal acidosis, no corresponding values for premature infants are available and no definite conclusions can be reached. It is, however, of interest to note that following the administration of NH_4Cl, the pH fell from 7·31 to 7·23 and the BE increased from −7·8 to −11·5. In spite of this acidosis the baby sucked well, was active and thrived during this period.

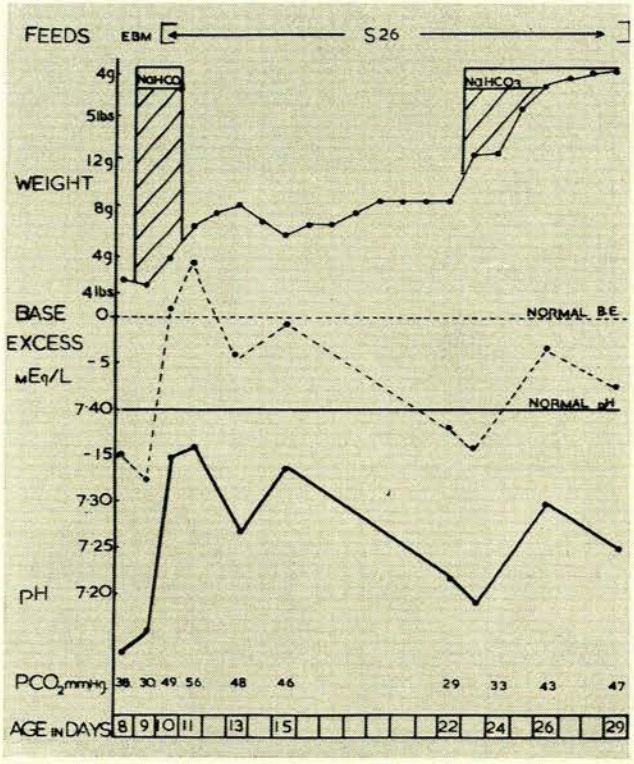


Fig. 5. See text.

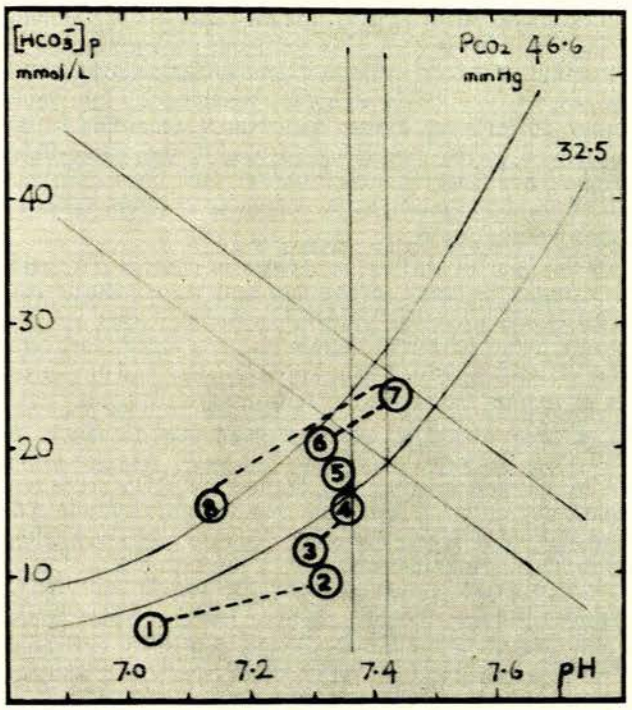
FACTORS WHICH MAY INFLUENCE THE AMOUNT OF BASE REQUIRED

The calculation from formulae of the amount of acid or base that can be given safely must only be regarded as a useful practical guide. The actual amount to be given, the speed of its administration and the need for repeating acid-base studies depend on a number of factors. They include the nature of the primary illness and the severity of the resulting associated pathophysical effects reflected in the acid-base status of the patient; the ability of the normal physiological mechanisms to deal with such derangements; and the response to therapy.

In addition, the possible effects of certain therapeutic procedures on acid-base metabolism must always be considered. These may have a specific or an indirect effect on the evolution and/or correction of metabolic acidosis. The 2 most common important procedures are peritoneal dialysis and exchange blood transfusion.

Effect of Peritoneal Dialysis

Patient J.K., aged 11 years, was admitted to hospital in a critical semi-comatose condition with renal failure. His blood urea was 535 mg./100 ml. and serum phosphate 19·5 mg./100 ml. (Fig. 6). A past history suggestive of possible obstructive congenital urinary disease was obtained.



No.	1	2	3	4	5	6	7	8
Date	13/7				14/7		15/7	21/7
Hours	0	11	14	20	33	39	58	
pH	7·04	7·32	7·31	7·35	7·34	7·34	7·44	7·16
PCO_2	25·0	17·0	22·5	30·5	33·0	37·5	36·6	43·4
BE	−19·0	−16·0	−14·0	−8·0	−7·0	−4·0	+0·7	−11·6
BB	21·0	30·0	30·0	35·2	38·0	46·0	48·0	34·0
Std. bicarb.	7·6	12·4	13·2	17·5	18·0	20·9	24·5	15·0
Act. bicarb.	6·8	8·8	11·0	16·2	18·0	20·0	24·2	15·2
Bicarb. therapy mEq.		174						
		← Dialysis 46 hours →						
Na^+	138	149			140		143	160
K^+	3·6	3·6			2·9		3·5	5·2
Cl^-	101	91			91		93	106
Urea	535	630			450		385	410
Ca^{++}	7·4	7·7			9·0			
HPO_4^{--}	19·5				17·3			
Alk. phos.	10·2							
Alb.		2·9			2·4		2·5	
Glob.		1·2			1·9		1·5	
Mg^{++}					2·1			

Fig. 6. Age of child 11 years; weight 28·5 kg. See text.

The standard regime for the management of renal failure was commenced. He was given 174 mEq. of sodium bicarbonate intravenously with some improvement in both his clinical condition and acid-base status (Fig. 6). Peritoneal dialysis was then commenced employing a 1·5% dextrose dialysis solution containing 140·5 mEq./l. sodium, 3·5 mEq./l. calcium, 1·5 mEq./l. magnesium, 101·0 mEq./l. chloride, 44·5 mEq./l. lactate and 15 G/l. of dextrose. To this solution potassium was added during the dialysis to correct the hypokalaemia (Fig. 6) which occurred during the dialysis. His progress and return to a normal acid-base status without the further administration of intravenous sodium bicarbonate is demonstrated in Fig. 6.

His clinical condition improved rapidly. Retrograde pyelography, however, demonstrated severe bilateral hydronephrosis, bilateral hydro-ureters and bladder neck obstruction. His condition was regarded as hopeless: he was left in a comfortable state and died 13 days later.

Effect of Exchange Blood Transfusion

Blood preserved by the addition of ACD (acid-citrate-dextrose) is commonly used for exchange transfusion. The pH of such freshly-drawn blood is about 7·0 and during storage may fall within 2 days to levels below 6·6.[12] This fall in pH is caused by an accumulation of hydrogen ions mainly from lactic and pyruvic acids produced by glycolysis in the red blood cells. Exchange transfusion with citrated blood may by itself result in a variety of mild or severe acid-base disturbances. This is determined to a large extent by the efficiency of (i) liver function and its ability to metabolize the trisodium citrate in the ACD solution to sodium bicarbonate; and (ii) the adaptation of the cardiovascular and respiratory systems to physiological changes which take place at birth. This includes the ability to cope with the effects of blood group sensitization, such as anaemia and congestive cardiac failure before the exchange transfusion, hazards of the immediate newborn such as IRDS, poor thermal control and the effects of exchange transfusion itself, such as volume overload and electrolyte disturbances, e.g. hyperkalaemia and hypocalcaemia.

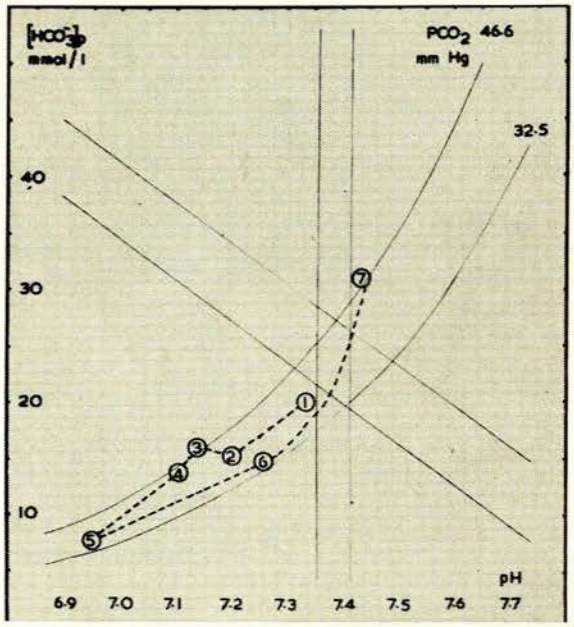
The adequate functioning of these systems is influenced by the general clinical state, age, gestational age and weight of the infant. Acid-base disturbances can be expected in premature infants, severely sensitized infants, or in those where exchange transfusion is carried out soon after birth, and before complete recovery from the stresses of labour and delivery.

The majority of exchange transfusions are fortunately not carried out in this latter group, but in moderately sensitized full-term infants or for hyperbilirubinaemia of prematurity in those who have had time for adaptation to their new environment. Full-term infants exchanged at the age of 12 - 24 hours, or premature infants exchanged after 3 days for hyperbilirubinaemia, commonly show a metabolic acidosis at the end of the transfusion which is followed by an overswing to a hypochloraemic metabolic alkalosis with the metabolic breakdown of citrate and release of HCO_3^-. Full-term infants transfused on account of hyperbilirubinaemia, from the second day onwards, usually demonstrate the metabolic acidosis only during the initial half of the exchange and at the end of the exchange the metabolic alkalosis is already present. Similar findings have been well documented by Calladine *et al.*[13] This hypochloraemic metabolic alkalosis requires no active treatment. It is corrected by normal renal function and by the chloride from milk feeds.

Acid-base disturbances of an acidotic nature may be life-threatening and pose a problem in correct management in severely affected anaemic, ill or premature infants requiring early exchange transfusion.[14] These disturbances present themselves in the following patterns:

Severe uncompensated metabolic acidosis. Infants are usually of less than 37 weeks' gestational age, are severely affected with anaemia and hepatosplenomegaly, and usually require exchange transfusion within the first 12 hours of life. They show evidence of metabolic acidosis even before the transfusion, but no disturbance of lung function. In them, correction of the metabolic acidosis does not take place immediately and it therefore becomes necessary to hasten the process by administering intravenous sodium bicarbonate. This must, however, be given with caution because the metabolism of the citrate in the transfused blood can ultimately supply the additional amount of base required. The difficulties which may arise are illustrated in the following case:

Infant R was delivered following a medical induction at 37 weeks' gestation. She weighed 2·9 kg. at birth, cord blood investigations revealed a positive Coombs test, total bilirubin was 4·1 mg./100 ml. and haemoglobin 7·6 G/100 ml. Her general condition was fair and an exchange with blood preserved with ACD was commenced at the age of 4½ hours. It was decided to abandon the procedure after 360 ml., before a full exchange (150 ml./kg.) had been carried out because of irregularity of the pulse. The pH and BE of the donor blood were 6·73 and –20 mEq. respectively. Umbilical arterial blood samples were taken during the exchange and up to the age of 28 hours (Fig. 7). The pH dropped from 7·34 to 7·13 and the BE decreased



No.	1	2	3	4	5	6	7
		1st exchange				2nd exchange	
Volume exchanged	0	100	300	360	0	510	
Hours	4½			6	16		28
pH	7·34	7·21	7·16	7·13	6·97	7·27	7·44
PCO_2	37·5	38·5	43·0	45·5	35·2	35·2	46·0
BE	–4·8	–13·5	–13·7	–13·8	–20·5	–9·7	+6·0
Actual bicarb.	19·2	14·8	15·0	14·8	8·0	15·7	30·5
Bicarb. therapy mEq.					4·0	7·0	7·0

Fig. 7. See text.

from –4·8 to –13·8 during the exchange. No correction of the metabolic acidosis took place and the infant's condition deteriorated clinically. A rise in the bilirubin at the age of 16 hours necessitated a second exchange transfusion. At this stage the arterial pH had fallen to 6·97 and the BE to –20·5. Four mEq. of sodium bicarbonate was given rapidly by intravenous injection and 7 mEq. was added to the donor blood (pH 6·84, BE –23) used during the second exchange of 510 ml. Following the exchange the infant showed improvement and a further 7 mEq. of sodium bicarbonate was given in a 10% dextrose drip into a scalp vein over 8 hours. By 28 hours an overswing to a metabolic alkalosis had taken place. The infant made an uneventful recovery.

It is important to note in this case that a large amount of base must have been obtained from the metabolism of the citrate, as the correction of a metabolic acidosis with a base excess of –20·5 in an infant weighing 2·9 kg. would require 36 mEq. of sodium bicarbonate according to the formula to correct the deficit of base in the extracellular space alone. Only 14 mEq. was in fact given and this proved to be too much, as the values at 28 hours indicated a metabolic alkalosis.

In the light of further experience in cases-at-risk, acid-base studies are now carried out and derangements corrected before exchange transfusion. Serial studies are performed following the procedure and any significant metabolic acidosis corrected with approximately half the quantity of sodium bicarbonate that is usually required.

Combined metabolic and respiratory acidosis. Some infants show the same clinical features as the previous group but in addition manifest the features of RDS such as a rapid respiratory rate, grunting, cyanosis and recession. The combination of severe haemolytic disease and severe clinical hyaline membrane disease in a premature infant is, in our experience, almost always fatal. Where the clinical hyaline membrane disease is mild or moderate, the outlook is improved. In these infants, therapy consists of correction of the metabolic acidosis with sodium bicarbonate and employment of the general principles of management of any respiratory acidosis.[15] Heparinized donor blood is used for the exchange transfusion whenever possible.

Uncompensated or partially compensated respiratory acidosis. These infants show no evidence of the respiratory distress syndrome though the respiratory rate may be above 70 and evidence of congestive cardiac failure may be present. Spontaneous correction of the respiratory acidosis usually takes place but resuscitation equipment, including a respirator for artificial ventilation, must be available next to the infant. During or after exchange transfusion, any evidence of respiratory failure, often heralded by apnoeic attacks, must be actively managed with assisted ventilation for a period of 12 - 24 hours.

SUMMARY

The development of micromethods for acid-base determinations is of particular interest to the paediatrician, as so many life-threatening illnesses affecting the paediatric age-group are associated with severe disturbances of acid-base metabolism.

The interpretation and application of acid-base investigations can be simplified by adopting the terminology used in this paper and by making use of methods which give a graphic representation of the acid-base status of the patient.

In the management of metabolic acid-base disturbances in a given disease, the clinician can with the help of formulae determine the amounts of acid or base which can be given with safety to the patient to correct a particular disturbance.

General principles in the management of metabolic disturbances have been discussed and illustrated with examples.

The management of respiratory acid-base disturbances will be discussed in a future publication.

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