Neonatal Acid-base Disturbances*

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The introduction of the micro-Astrup technique (Siggaard Andersen, Engel, Jørgensen, and Astrup, 1960) with the subsequent modification of the nomogram (Siggaard Andersen, 1962) has given a rapid and simple method for separating the respiratory and metabolic components of acid-base equilibrium in the blood. Arterialized capillary blood from a heel stab has been used for the estimation of pH and for equilibration with oxygen/carbon dioxide mixtures of known composition. For the present discussion the respiratory aspects will not be considered, but only the so-called metabolic changes and the parameter used is the base excess. Base excess is defined as zero for blood with a pH of 7.40 at a PCO_2 of 40 mm. Hg. A negative base excess indicates a deficit of base or an excess of fixed acid, i.e. a metabolic acidosis. Positive base excess indicates an excess of base or a deficit of fixed acid, i.e. a metabolic alkalosis. The normal range for base excess is from -2 to $+2$ mEq/l.

Metabolic Acidosis

Case 1. A 3-day-old male infant (G.W.), 2.78 kg., was admitted with a diagnosis of oesophageal atresia and tracheo-oesophageal fistula. This baby had been at home for 2 days after a normal pregnancy and was admitted to another hospital with a history suggestive of fits. At first these fits were thought to be due to hypoglycaemia, and the baby was hypothermic with a rectal temperature of 89.8° F. (32° C.). The blood sugar was 11 mg./100 ml., and oral glucose was given. The baby then became cyanosed and the diagnosis of oesophageal atresia was made after radiological examination.

On admission to our unit the baby's temperature was normal but sclerema was spreading up from lower limbs on to buttocks, and there were widespread pulmonary changes. The Astrup measurements revealed a metabolic acidosis with a base excess of -10.8 mEq/l. That night the tracheo-oesophageal fistula was divided, but because of the poor general condition no attempt was made to mobilize the segments sufficiently to make a primary anastomosis. No intravenous fluids were given. The metabolic acidosis increased, the base excess rising

to -15.2 mEq/l. At this point a gastrostomy was made, and the baby was fed with dextrose and then milk in steadily increasing quantities. The base excess thereafter steadily decreased (Fig. 1). The acidosis increased again only once when the baby's temperature was allowed to drop below 98° F. (37° C.). The sclerema neonatorum became generalized within 12 hours of admission and remained so for 48 hours. It then resolved completely. Penicillin and vitamin K were the only drugs given, apart from the anaesthetic agents.

Case 2. On admission the second infant (C.C.) was 36 hours old, weighed 2.98 kg., and had a metabolic acidosis with a base excess of -8.2 mEq/l. Operation was performed soon after admission and the tracheo-oesophageal fistula was divided, followed by primary anastomosis of the oesophagus. No intravenous fluids were given; blood loss was estimated at not more than 10 ml. and therefore none was transfused. Feeding was begun by a transanastomotic plastic tube 24 hours after

FIG. 1.—Metabolic acidosis in a newborn male infant with oesophageal atresia and tracheo-oesophageal fistula. Gastrostomy was carried out at operation.

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FIG. 2.—Metabolic acidosis in a newborn male infant with oesophageal atresia and tracheo-oesophageal fistula. Primary anastomosis was done.

operation, and a rapid correction of the metabolic acidosis resulted (Fig. 2).

Comment. The cause of metabolic acidosis in association with oesophageal atresia is uncertain, but

c.c. two of the factors that will contribute to it are the intermittent hypoxia and the fasting that these babies undergo. That the latter is not the only factor is suggested by the work of Beard, Panos, Burroughs, William, and Russell (1961), but unfortunately they did not report all the results of their studies on neonatal fasting. The classic studies of Gamble, Ross, and Tidsall (1923) demonstrated that fasting resulted in metabolic acidosis. In the 5 adults we have studied recently, a metabolic acidosis has always accompanied starvation and has been rapidly corrected by refeeding. The extent of the changes in base excess in a fasting adult are shown (Fig. 3).

Whether metabolic acidosis of the degree described has harmful effects in a newborn infant is not known. Further, the administration of bicarbonate or THAM alters only the end-result of the metabolic processes, so that it is preferable to focus attention on the primary defects—hypoxia and starvation—and to treat them, rather than to become obsessed with correction of the blood chemistry which may in itself not be deleterious.

Metabolic Alkalosis

Case 3. A 14-day-old baby whose vomiting was thought to be due to a cerebral birth injury, but who had a complete obstruction of the second part of the duodenum proximal to the ampulla of Vater, was admitted in a moribund condition. Weight at birth had been 3.84 kg. and was now 2.42 kg., a weight loss of 37%. Not surprisingly this baby had a metabolic alkalosis, and on admission the serum concentration of sodium was 157 mEq/l., potassium 10 mEq/l., chloride 77 mEq/l., the total CO_2 was 50 mEq/l., and urea approximately 300 mg./100 ml. After the intravenous administration of 200 ml. normal saline and 100 ml. half-normal saline with 2.5% dextrose, during 15 hours, a duodenojejunostomy was made. The metabolic alkalosis decreased 12 hours after operation, but in 30 hours had returned to its former level. This apparent correction may simply have been the result of a hidden metabolic acidosis from the hypoxia and poor tissue perfusion after operation. Twenty-four hours after operation feeds of 5 ml. breast milk hourly were begun through a tube passed across the anastomosis into the jejunum, and the gastric aspirate also was given. The metabolic alkalosis persisted, and so 6 mEq NH_4Cl were given over 6 hours. Coincident with the administration of the NH_4Cl was a marked improvement in the baby's condition and subsequently the remaining alkalosis slowly subsided (Fig. 4).

Comment. Ammonium chloride has been out of favour since the work of Gamble and Ross (1924). However, their studies demonstrated only that ammonium chloride alone would not correct a deficiency of anion and cation, i.e. that if sodium was deficient it had to be replaced. As a result the

FIG. 3.—Metabolic acidosis in a normal fasting woman.

FIG. 4.—Metabolic alkalosis in a newborn male infant with complete duodenal obstruction.

importance of the chloride ion was obscured for many years, but recently its importance has been clearly demonstrated and stressed by Goodwin and Oakley (1965), Kassirer, Berkman, Lawrenz, and Schwartz (1965), de Graeff, Struyvenberg, and Lameijer (1964), and Lemieux and Gervais (1964). The chloride ion has an effect on the renal tubules and lowers the bicarbonate threshold, allowing correction of the alkalosis.

Seldom is it necessary to give ammonium chloride, and in most situations sodium chloride or potassium chloride will supply sufficient of the anion. Certainly babies with a base excess of $+2$ mEq/l. do not need ammonium chloride as recommended by Kildeberg (1964).

In summary I would stress that more attention should be paid to the aetiology of metabolic acidosis. The institution of early feeding, if necessary by tailoring of our surgery to allow this, is of more importance than the administration of THAM or bicarbonate. In metabolic alkalosis too much attention has been focused on the cations, but the anions, particularly chloride, are of considerable importance.

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