Acid-base Determinations in Normal Premature Infants in the First Two Months of Life

A. F. MALAN,* A. EVANS, and H. DE V. HEES

From the Neonatal Respiratory Unit, Department of Child Health, University of Cape Town, South Africa

In a previous report from this unit (Malan, Evans, and Heese, 1965) it was shown that in the first 72 hours of life there are several differences in acid-base status between premature and full-term infants. While term infants approached normal levels for adults, the pattern in premature babies was unstable, with a tendency towards acidosis. Metabolic (non-respiratory) acidosis has been recorded in premature infants up to the age of 70 days (Reardon, Graham, Wilson, Baumann, Tsao, and Murayama, 1950; Blystad, 1956; Alvarez de los Cobos, Jurado-García, Sagaón, and Leon, 1957; Ranlov and Sigggaard Andersen, 1965).

The present study reports acid-base determinations in premature infants between the 4th and 60th days of life and is designed to serve as an addendum to the rather scanty publications on this age period.

Material and Methods
An unselected group of premature infants (birthweight below 2.5 kg.) was studied in the Peninsula and Groote Schuur maternity hospitals in Cape Town. Only normal healthy babies who were gaining weight were investigated. Most of the babies were fed on full strength cows’ milk formulae, though a few were given expressed breast milk for the first 7 to 14 days.

There were 50 patients in the series and 149 acid-base determinations were made on them between the ages of 3 and 60 days. The infants were grouped according to gestational age as calculated from the date of the mother’s last menstrual period. These groups were as follows.

Group A: babies of less than 34 weeks’ gestation—20.
Group B: babies of gestation 34-36 weeks—19
Group C: babies of gestation over 36 weeks—11.

Arterialized capillary blood was collected and acid-base values determined by means of the Astrup method as previously described (Malan et al., 1965).

Results
The values for pH, Pco₂, base excess, buffer base, standard bicarbonate, and actual bicarbonate are set out in Table I. The results are grouped into 5 time periods. There seemed to be no appreciable difference in values from the 4th to the 8th week.

The mean values for pH, base excess, buffer base, and standard bicarbonate show a gradual movement in the direction of metabolic acidosis from the 4th to the 14th day. During the following (3rd) week they remain stationary and thereafter show a rise in values. On the other hand, the mean Pco₂ shows little change.

Although there is a wide spread of acid-base values, the majority of the babies had a metabolic acidosis, usually uncompensated, and in some cases rather quite severe. Several infants presented with combined metabolic and respiratory acidosis.

No consistent difference was found in the values for the babies in the two most immature groups (A and B). Those in Group C (maturity over 36 weeks) however, showed consistently higher pH and lower base deficit values, especially during the second month (Table II).

Discussion
The values recorded in this study are similar to those of other authors. In all reports the pH is low. The Pco₂, in contrast with the low values found in the first few days of life, is close to the adult norm of 40 mm. Hg. In the second month of life Ranlov and Sigggaard Andersen (1965) found the value to be slightly higher (44 mm. Hg). The corresponding figure in this study was 42.1 mm. Hg. Some of our patients also showed the occasional high Pco₂ levels which were found by Kildeberg (1964a) in apparently well premature infants. The base deficit in our patients was lower than that reported by Blystad (1956), Kildeberg (1964a), and Ranlov and Sigggaard Andersen (1965), but compares well with the figure calculated from the data of Reardon et al. (1950).

The development of metabolic acidosis after 48-72 hours of age had been previously suspected (Malan et al., 1965) and is confirmed in this study.
over-all course in our patients was found to be identical with that described by Ranlov and Siggaard Andersen (1965). The onset of the acidosis seems to us to coincide with higher protein loads in the infants' feeds. A relation between metabolic acidosis and a high protein diet, described by Darrow, Da Silva, and Stevenson (1945), has been discussed in several recent reports (Kildeberg, 1964b; Ranlov and Siggaard Andersen, 1965). The metabolic acidosis disappeared by the end of the third week according to Ranlov and Siggaard Andersen (1965). This was not so in our patients. Our data were not analysed in terms of late metabolic acidosis, as has been done by Kildeberg (1964b) and Ranlov and Siggaard Andersen (1965), and though all our infants were gaining weight at the time of sampling, many must inevitably fall into that category.

The relation between maturity and the degree of metabolic acidosis is controversial. Alvarez de los Cobos et al. (1957) and Ranlov and Siggaard Andersen (1965) both recorded a direct correlation between low birthweight and metabolic acidosis. On the other hand, neither Reardon et al. (1950) nor Kildeberg (1964b) found any such correlation. In this study a significant difference was only detectable when infants of less than 36 weeks' gestation were compared with those of a greater degree of maturity.

**Summary**

Acid-base values for 50 premature infants aged between 3 and 60 days are presented. From the 4th to the 14th day there was a gradual development of uncompensated metabolic acidosis which slowly improved after the 3rd week of life. Despite the acidosis, all the infants were well and gaining weight at the time of determination.

The metabolic acidosis is increased in infants of less than 36 weeks' gestation.

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