BIOCHEMISTRY OF FOETAL ASPHYXIA AND POTASSIUM DEPLETION IN CHRONIC FOETAL ASPHYXIA

by

HARINDER AMARJEET SINGH M.B., B.S. (Punjab, India).

Being a Thesis
presented for the
degree of

DOCTOR OF MEDICINE

at the

University of Adelaide,
Department of Obstetrics and Gynaecology

March 1971.

CONTENTS

SUMMARY.

The present study comprises of acid-base and electrolyte balance along with lactate and pyruvate levels of maternal and umbilical cord blood from 141 mothers and their infants at delivery. The new-born infants at birth had a certain degree of respiratory and metabolic acidosis, the latter being more marked in the asphyxiated infants. The pCO_2, plasma sodium, potassium, haematocrit, whole blood potassium and lactic acid levels in cord arterial blood were higher whereas pH, standard bicarbonate, red cell potassium and pyruvic acid were lower than the maternal blood. Foeto-maternal correlations were obtained for all the constituents and their statistically significant relations have been mentioned wherever appropriate.

Inter-relationship of acid-base and electrolyte balance has been studied in normal and asphyxiated infants. The red cell potassium level showed significant positive correlations with acid-base parameters and negative correlation with plasma potassium value in cord arterial blood. Metabolic acidosis was more pronounced with low concentration of red cell potassium in infants with signs of chronic asphyxia. It was established that potassium level in whole blood was related to potassium

level in red cells and not plasma. Red cell potassium is a good index of body potassium level and the acid-base status of the foetus.

Apgar of the infant at 1 minute showed significant relation to acid-base values but not with electrolyte values.

The changes in acid-base and electrolyte balance in 19 new-born infants were followed by repeated sampling of umbilical arterial blood during the first 3 hours after birth. The acidosis at birth was mainly metabolic in nature but also temporarily of respiratory type, as the pCO_2 tension fell rapidly during the first half-an-hour, whereas the standard bicarbonate level decreased and lactic acid level increased during this time. The plasma potassium level gradually decreased with a slight rise at 2 hours of age and the red cell potassium level gradually increased with a slight fall at 1 hour of age. These levels at 3 hours had not reached the normal adult levels. Infants were grouped according to pH of their cord arterial blood at birth into 'low pH' ($\leq$7.20) and 'normal pH' ($\geq$7.20) groups. A significant difference in the mean values of pH, pCO_2, standard bicarbonate, base deficit, red cell potassium, lactic acid, pyruvic acid and lactate/pyruvate ratio was obtained in the two groups.

Infants with 'low pH' at birth showed marked difference during the recovery from acidosis compared with group of infants with 'normal pH'. The infants born with chronic asphyxia took longer to recover and even after three hours and despite the treatment these infants had high lactic and pyruvic acid and low red cell potassium levels. This low red cell potassium could be a reflection of total body potassium depletion which results from chronic asphyxia.

Electrocardiograms were recorded on these infants approximately one hour after birth. The electrocardiographic findings were not consistent with the acid-base values or blood potassium levels. Acidosis was associated with decrease in red cell potassium with or without changes in plasma potassium. In 4 infants the low plasma and red cell potassium concentrations were associated with electrocardiographic findings of hypopotassaemia in adults.

The biochemical estimations done in 21 scalp blood samples showed normal acid-base and red cell potassium values. The mean lactic acid concentration was higher in the scalp blood than maternal venous blood before delivery. The lactic acid level in scalp blood was related to the Apgar of the infant at 1 minute after birth.

In nine cases cord and membrane tissue potassium values were estimated. The mean potassium levels were higher in these tissues in infants with low cord arterial pH.

The present study confirms the hypothesis that during acidosis of the new-born the shift of potassium into plasma is from the red blood cells. Significant linear correlation of red cell potassium to acid-base and plasma potassium concentration in the foetus has been demonstrated.

A hypothesis is advanced that decreased red cell potassium is a result of total body potassium depletion. This hypothesis can be confirmed only by measuring the total body potassium content in the new-born infants.