impaired in the premature infant. Preoccupation with maintenance of biochemical homeostasis in the treatment of prematures and renewed interest in tryptophan, tyrosine and phenylalanine metabolism by the newborn prompted us to this study of urinary excretion of tryptophan and tyrosine metabolites by 11 full-term and 12 premature infants on their 1st day of life and 9 full-term and 11 prematures on their 15th day of life. Metabolites from all three main catabolic pathways of tryptophan were detected in both groups of infants. The main differences between full-term and prematures were a) 5-indole-propionic acid was found only in the urine of full-term infants on the first day of life; b) 3-indoleacetic acid was not found on the 15th day of life in the urine of full-term infants; and c) on the first day of life the urine of 10–20 % of prematures contained 5-OH-tryptophan, indoxyl-acetyl-glutamine and 5-OH-anthralinic acid. On the 15th day of life the number of metabolic products was decreased, probably because tryptophan was now incorporated in proteins.

Twenty phenolic acids were observed in both groups of infants, but their excretion varied a great deal. Homogentisic acid was excreted in the urine of half of the full-term infants on the 15th day of life, but not on the 1st day of life. The appearance of this acid in the urine of some full-term infants on the 15th day of life is suggestive of the maturation of the enzymes functioning in the main metabolic pathway of tyrosine via homogentisic acid.

The 24-hour urinary metacatecholamines and vanilmandelic acid (VMA) were also determined in the same premature and full-term infants. Variations of excretion of VMA generally followed those of metacatecholamines, but the excretion of VMA by the prematures on the 15th day of life was 4–5 fold that of the 1st day. In contrast, a moderately decreased VMA excretion was noted in the urine of full-term infants.

We are inclined to believe that the observed differences in metacholamine and VMA excretion are due to the delayed maturation of 3-O-methyltransferase and monoaminoxidase, the enzymes responsible for the metabolism of catecholamines to VMA. But the marked increase in VMA excretion on the 15th day by the prematures has to be attributed to an 'overmaturation' of the last enzyme or to other factors.

37 *Acid-Base and Electrolyte Changes During Exchange Transfusion in the Newborn.* Francesca Severi, C. Belloni and G. Rondini, Pediatric Clinic of the University of Pavia, Italy.

Twenty-six newborn infants (eighteen full-term infants and eight prematures) requiring exchange transfusion have been included in this investigation. Citrated blood was used. 1 ml of calcium gluconate was added to every 100 ml of blood in 9 full-term infants and in all the prematures; 3 ml of calcium gluconate were added in 9 full-term infants. The exchange transfusion lasted in every case about 2 hours.

The blood samples for plasma Na, K, Ca, bilirubin and total protein levels were taken from the umbilical vein before the transfusion, during its course and 1, 2, 3 and 24 hours after the end of the transfusion. At the same time, but also once daily up to the 5th day, pH, pCO_2, base excess and standard bicarbonate were measured with the micromethod of Astrup in the arterialized capillary blood.

The mean values show that in full-term newborns the exchange transfusion caused a continuous rise of the pH which became more evident after the first half an hour; the highest value was reached 2–3 hours after the end of the transfusion. The pH gradually decreased during the following 5 days. The base excess and the standard bicarbonate values, after a slight initial reduction, rose during the transfusion and afterwards, up to the 2nd-24th hour and gradually decreased in the following days. Only slight differences have been observed between the group treated with 1 ml of calcium gluconate to every 100 ml of blood and that treated with 3 ml.

In premature infants, a slight initial fall of the pH values was observed; afterwards the rise of the pH and the tendency to metabolic alcalosis was slower and less evident than in full-term newborns. Neither in full-term newborns nor in prematures was observed serious metabolic acidosis caused by exchange transfusion.

K and total protein plasma levels showed a slight reduction in almost every case during exchange transfusion; bilirubin decreased constantly; Ca plasma levels increased almost in all cases. Na plasma levels showed only slight variations.

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