infants may need a higher insulin level to effectively reduce hepatic endogenous glucose production or enhance glucose disposal or both. The possibility of a smaller insulin-sensitive tissue mass in LBW infants may be another factor that needs to be explored. In particular, adipose tissue mass is very small in very immature neonates.

The data on urine volume and glucose excretion are of interest in that an osmotic diuresis was not observed, despite a significant increase in serum and urine glucose levels. The net effect is apparent since the tubular water conservation was achieved, probably because the total amount of glucose being excreted constituted an osmolar load that was too small to produce an osmotic diuresis.[11]

From the clinical standpoint, although 20 gm/kg/day of glucose intake did not result in an osmotic diuresis, hyperglycemia was evident and its effect on brain metabolism is unknown. In Rhesus monkeys, Myers[12,13] has shown that hyperglycemia may result in significant brain injury when asphyxia is superimposed.

REFERENCES

1. Dweck HS, Cassady G: Glucose intolerance in infants of very low birth weight: I. Incidence of hyperglycemia in infants of birth weights 1,100 grams or less. *Pediatrics* 53:189, 1974.
2. Zarif M, Pildes R, Vidyasagar D: Insulin and growth-hormone responses in neonatal hyperglycemia. *Diabetes* 25:428, 1976.
3. Dweck HS, Siegal A, Cassady G: Glucose intolerance of infants of very low birth weight, abstracted. *Pediatr Res* 6:149, 1972.
4. Cser A, Milner RDG: Glucose tolerance and insulin secretion in very small babies. *Acta Paediatr Scand* 64:457, 1975.
5. Salle B, Ruitton-Uglienco A: Glucose disappearance rate, insulin response and growth hormone response in the small for gestational age and premature infants of very low birth weight. *Biol Neonate* 29:1, 1976.
6. Darki A, Fiser RH Jr, Arrington RW, Beard AG: Glucose homeostasis in the very low birth weight (LBW) infant, abstracted. *Clin Res* 25:79A, 1977.
7. Dweck HS, Brans YW, Sumner JE, Cassady G: Glucose intolerance in infants of very low birth weight: II. Intravenous glucose tolerance tests in infants of birth weights 500-1380 grams. *Biol Neonate* 30:261, 1976.
8. Cornblath M, Schwartz R: *Disorders of Carbohydrate Metabolism in Infancy*, ed 2. Philadelphia, WB Saunders Co, 1976, p 218.
9. Cowett RM, Susa JB, Oh W, Schwartz R: Endogenous glucose production during constant glucose infusion in the newborn lamb. *Pediatr Res*, to be published.
10. Bowie MD, Mulligan PB, Schwartz R: Intravenous glucose tolerance in the normal newborn infant: The effects of a double dose of glucose and insulin. *Pediatrics* 31:590, 1963.
11. Pratt EL, Snyderman SE: Renal water requirement of infants fed evaporated milk with and without added carbohydrate. *Pediatrics* 11:65, 1953.
12. Myers RE: Anoxic brain pathology and blood glucose, abstracted. *Neurology* 26:345, 1976.
13. Myers RE, Yamaguchi M: Effects of serum glucose concentration on brain response to circulatory arrest, abstracted. *J Neuropathol Exp Neurol* 35:301, 1976.

ACKNOWLEDGMENT

We are grateful to Ms. Jackie Beaudet, Mrs. Barbara Davis, and Mrs. Kathleen Petzold for their technical and laboratory assistance in performing the studies.

CORRECTION

An error was made in the article "Vitamin E and Neonatal Hemolysis" by Gross et al. (*Pediatrics* 59[suppl]:995, June 1977). In "Materials and Methods" on page 995, it states that infants received tocopherol acetate. The infants actually received the free alcohol form of tocopherol.

Vitamin E and Neonatal Hemolysis

Steven J. Gross, Stephen A. Landaw and Frank A. Oski

Pediatrics 1978;61;549

Vitamin E and Neonatal Hemolysis

Steven J. Gross, Stephen A. Landaw and Frank A. Oski

Pediatrics 1978;61;549