maintained in a 'neutral' thermal environment from ages 4 to 140 hours. Infants were fasted till after the 12–20 hour sample when feedings were begun. Mean values (±SD):

Age	pH	$PaCO_2$	BE
4–6	7.32 (0.01)	38 (1.00)	–5.6 (0.6)
12–20	7.34 (0.10)	34 (1.60)	–6.1 (0.5)
24–30	7.37 (0.01)	32 (0.90)	–5.6 (0.4)
36–42	7.39 (0.02)	32 (1.67)	–5.1 (0.4)
Age	FFA	Ketones	Glucose
4–6	0.99 (0.16)	0.7 (0.23)	48.4 (4.1)
12–20	1.32 (0.12)	0.87 (0.12)	60.0 (5.3)
24–30	1.29 (0.12)	1.12 (0.21)	55.9 (9.3)
36–42	0.90 (0.08)	0.85 (0.15)	62.8 (3.4)

A significant increase in FFA ($p < 0.001$) and ketones ($p < 0.05$) occurred in fasting infants at age 12–18 h despite an increase in glucose ($p < 0.001$). Linear correlation existed between ketones and FFA ($y = 3.4$ [FFA]$+0.7$; $r = 0.48$; $p < 0.01$). FFA varied independently of glucose. Acid-base parameters bore no correlation to the energy substrates. FFA and ketones fell to initial levels by 36–42 h; glucose remained at 62 to 67 mgms % to age 6 days. The data indicates that premature infants at neutral temperature and in a normal acid-base state rely on lipid substrates during the first 5 days of life despite adequate blood glucose concentration. (SPR)

77 *Ketotic Hypoglycemia: The Effect of Beta-Hydroxy Butyrate (β-OHB) Infusion and Heparin-induced Ketosis.* HENRY S. SAULS*, Univ. of Minn. Med. Sch., Minneapolis, Minn. (introduced by Robert A. Ulstrom).

Cause and effect are implied in the term 'ketotic hypoglycemia' used by COLLE and ULSTROM (J. Ped. *64:* 632 [1964]) to describe a childhood syndrome of hypoglycemic convulsions associated with ketonuria. MADISON (J. clin. Invest. *43:* 408 [1963]) showed a stimulatory effect of ketones on insulin secretion and postulated this as a feedback mechanism to prevent fatal ketosis. Two techniques to induce ketosis were used to study patients with ketotic hypoglycemia: 1. β-OHB was infused intravenously during a 2 to 6 hour period; 2. Heparin was injected intramuscularly to promote lipolysis. Patients fasted throughout the test period. The response of patients and control subjects was compared to a control day when no heparin was given. Blood sugar (BS), immunoreactive insulin (IRI), non-esterified fatty acids (NEFA) and ketones were measured. β-OHB caused a slow decline in BS at a rate more rapid than with starvation alone. Insignificant increases in IRI ($\bar{x}9$ μU) within 60 minutes were seen in 3 patients whereas the values of a control subject rose 65 μU within 20 minutes. Patient's values remained low throughout the test period in spite of progressively increasing ketosis. After the initial rise and fall, IRI slowly increased in the control subject at the 4th, 5th and 6th hours of infusion.

A 16-hour fast did not cause hypoglycemia in patients or control subjects. With heparin, NEFA and ketones increased to high levels. Three of 4 patients became hypoglycemic by the 16th hour ($\bar{x}$ BS 21 mg %). Failure of two patients to respond to i.v. glucagon suggests depleted liver glycogen. IRI levels were low throughout the test periods. Hypoglycemia may be due to failure of ketone stimulation of insulin production with concomitant preservation of liver glycogen. (APS).

78 *Galactose-1-Phosphate Accumulation in Red Cells of a Neonate.* RUTH C. HARRIS and STANLEY V. ISBELL*, Columbia Univ., College of Physicians and Surgeons, New York, N.Y.

SCHWARTZ, HOLZEL and KOMROWER have demonstrated high levels of galactose-1-phosphate in tissues of newborns deficient in galactose-1-phosphate uridyl transferase activity. Following their recommendations to maintain a known heterozygotic pregnant mother on a galactose free diet, we examined the cord blood of a galactosemic infant. The levels of galactose-1-phosphate were found to be approximately four times normal. At age of ten days, the infant's red cell content of galactose-1-phosphate had increased to seven times normal, even though he was on a galactose free diet. Over the ensuing five weeks, the levels slightly decreased. Intravenous administration of galactose-1-phosphate uridyl transferase in 35 ml of packed normal red cells resulted in prompt disappearance of galactose-1-phosphate and decrease in liver size. Studies of maternal galactose levels in another pregnant woman as well as evaluation of maternal and newborn lactation are in process. (SPR)

79 *Prolonged Infusion of the Small Intestine of the Rat with Dilute Solutions of Lactic Acid.* J. RICHARD HAMILTON*, Dept. Ped., Univ. Toronto, Res. Institute, Hosp. for Sick Children, Toronto, Canada (introduced by Donald Fraser).

Using a new technique for the continual infusion of solutions into the jejunum of small animals, dilute solutions of lactic acid (2.0g %–3.5 g %) were infused into the rat for 10 days and their effect on fat absorption and mucosal structure assessed. Thirty-five rats were studied in 5 groups. Control rats (water infused) ate well, gained weight, excreted normal amounts of fat (2.9± 1.9 %) and showed no abnormality of mucosal structure (villous length 465±35 μ). Although dilute acid (2.0 g %) produced no abnormality, groups infused with more concentrated acid did show significant increases in fat excretion (Lactic acid 2.5 %–6.5±2.1 %; 3.0 g %–7.2±2.1 %). Lesions of the mucosa were found localized to the site of infusion in both of these groups, characterized by shortened villi (2.5 g %–276±95 μ, 3.0 g %–266±56 μ) and some epithelial cell derangement at the villous tips. In some rats, fusiform extrusions of epithelial cells were seen at villous tips indicating abnormal turnover of these cells. Rats infused with 3.5 % lactic acid died within 72 hours from intestinal perforation at the infusion site. The findings suggest that the effects observed were due directly to the acid infusions and not secondary to factors such as undernutrition or altered intestinal microflora. The observations made may not be specific for lactic acid, but they do demonstrate that intraluminal factors may impair fat absorption and mucosal structure. In this experiment, the particular factor used is an organic acid that is present in the human intestinal lumen. The infusion technique may be a valuable experimental model for future studies of the malabsorptive state. (SPR)

80 *Stimulation of Active Intestinal Transport by Actinomycin D.* CONSTANTINE S. ANAST, LUCILLE F. ADAMSON* and JOAN A. FOLWELL*, Univ. of Mo. Sch. of Med., Columbia, Mo., and Harvard Med. Sch., Boston, Mass.

It is well known that actinomycin D, an inhibitor of DNA directed RNA synthesis, alters enzyme activity.