effect on Ca^{++}. The clinical significance of this observation requires further evaluation.

Controlled clinical trial of phenobarbital (PB) and light for management of neonatal hyperbilirubinemia in a predominant Negro population. O. S. VALDES, H. M. MAURER, C. N. SHUMWAY, D. DRAPER, and A. HOSSAINI. *Med. Coll. of Virginia, Richmond, Va.* (Intr. by W. E. Laupus).

Low birth weight infants, less than 24 hrs of age, were randomly assigned to receive either oral PB, 5 mg/kg/day for 5 days, blue light (200–300 footcandles) continuously for 4 days, a combination of PB and light, or no treatment. 90% or more of the infants in each group were Negroes. Infants with a positive Coomb's test were excluded.

	No.	Total serum bilirubin concentration, mg% (mean ± S.E.)					
		day 1	day 2	day 3	day 4	day 5	day 6
Control	15	2.4±0.5	5.0±0.7	8.0±0.8	8.2±0.8	8.0±1.0	6.7±1.2
PB	23	2.4±0.4	4.0±0.5	5.1±0.6	5.5±0.7	5.6±0.7	4.7±0.8
Light	19	1.9±0.5	3.1±0.6	3.4±0.7	3.0±0.7	3.4±0.8	2.9±1.0
PB + light	18	2.0±0.5	3.3±0.6	3.6±0.7	3.7±0.8	3.4±0.9	3.2±0.9

By the 3rd day, infants receiving PB, light, and PB and light had significantly lower mean bilirubin concentrations than the controls. Those receiving light had the lowest values. Combining PB with light did not have an additive effect. Bilirubin concentrations of 10 mg% or higher were found in 66% of the controls, 26% of the PB group and in none of the other 2 groups. Mild lethargy occurred in 5 infants receiving PB and mild diarrhea occurred in 2 receiving light. The findings indicate that in Negroes continuous phototherapy is more effective than PB in lowering the serum bilirubin concentration. Results are not improved by combined therapy. PB may be useful when phototherapy is not available.

Hepatic metabolism and transport of bilirubin during physiologic jaundice in the newborn rhesus monkey. LAWRENCE M. GARTNER and DONNA LANE. *Albert Einstein Coll. of Med., N. Y., N. Y.*

Maximal hepatic uptake, conjugation and excretion of bilirubin was studied simultaneously in 12 newborn rhesus monkeys ranging in age from 4 hours to 19 days. In this model of human physiologic jaundice, maximum serum bilirubin concentrations prior to infusion of bilirubin were 4.5 mg/100 ml between 12 and 36 hours of life and less than 1.0 mg/100 ml by 48 hours. Endogenous bilirubin excretion in bile in monkeys 12 hours to 19 days of age was 2 to 10 times greater than in normal adult rhesus monkeys. During the first 36 hours of life maximal cumulative hepatic uptake of bilirubin was 35% of adult capacity; hepatic conjugation of bilirubin *in vitro* and *in vivo* was 5% of adult capacity; and hepatic excretory capacity was 10% of adult capacity. Each of these functions increased rapidly to adult levels by the fourth day of life. The rate-limiting step in the transfer of bilirubin from blood to bile at 12 to 36 hours of age is the capacity to conjugate bilirubin with glucuronic acid. In the period immediately following this, significant accumulation of direct-reacting bilirubin occurs in liver and sera during infusion of unconjugated bilirubin, indicating that excretion is the rate-limiting process after 36 hours of age. These studies suggest that physiologic jaundice results from a markedly increased load of bilirubin presented to the liver either from increased bilirubin synthesis or enhanced intestinal reabsorbtion and marked limitation in the capacity to conjugate.

Studies performed at 4 hours of age in a monky born 2 weeks post-maturely revealed that hepatic transport and conjugation of bilirubin was fully mature, indicating that maturation of each of these functions may occur in utero.

A controlled study of intravenous fibrin hydrolysate supplement in prematures <1.3 kg. M. HEATHER BRYAN, PATRICK WEI, RICHARD HAMILTON, SANFORD H. JACKSON, INGEBORG C. RADDE, GRAHAM W. CHANCE, and PAUL R. SWYER. *Univ. of Toronto and The Research Inst., The Hosp. for Sick Children, Toronto, Ont. Canada.*

We have compared the effect of early supplemental intravenous 3.5% fibrin hydrolysate plus 10% dextrose to 10% dextrose alone on the mortality, morbidity, weight gain and biochemical changes in 2 equal groups of 15 low birth weight infants of appropriate gestation. The initial alimentation was intravenous with gradual replacement by oral formula over the first 2 weeks of life. Total fluid intake of 200 ml/kg/day was maintained. Total caloric intake and urinary output did not differ between groups. The amino acid infants received twice the amount of protein (g/kg/day) as the controls. Mortality did not differ between groups when infants were assessed by body weight. In the survivors apnoea was significantly less frequent in those receiving amino acid and regain of birth weight more rapid. Total protein and BUN were higher in those on amino acid reflecting increased N_2 intake, and serum PO_4 lower. Serum electrolytes, sugar, osmolality, HCO_3 did not differ. Fatal cases in the amino acid group, many <1.0 kg, exhibited an excessive rise in BUN, and high plasma osmolality within 3 days of infusion.

	Cals/kg/day		Mortality		Regain of birthweight in survivors
	oral	IV	1.0–1.3 kg	<1.0 kg	
Amino acid	79	30	2/8	5/7	14 days
Control	84	21	2/12	2/3	22

Fetal-maternal metabolic fuel adaptations to caloric deprivation in human pregnancy. YOUNG J. KIM, VINCENT LYNCH, and PHILIP FELIG. *Yale Univ. Sch. of Med., New Haven, Conn.* (Intr. by Charles D. Cook).

Although glucose is considered the sole metabolic fuel utilized by the fetus to meet its energy requirements, induction of ketone-oxidizing enzymes in fetal brain tissue has recently been demonstrated in experimental animals fasted during pregnancy. The significance of such enzyme induction to fetal fuel economy is dependent ultimately on substrate availability. To examine the latter during caloric deprivation in human pregnancy, 14 physically healthy pregnant women (PW) undergoing theraputic abortion for pychiatric reasons during week 16–22 of gestation and 6 non-pregnant women (NP) were fasted for 84 hours. In PW, blood β-OH-butyrate and acetoacetate rose to 4.2 and 0.9 mM/L respectively, and were 2–3 fold higher than in NP for the first 60 hours of the fast. Throughout the fast plasma glucose was significantly lower ($P < .005$) in PW falling to 47 ± 1 mg%, 25% below NP levels. Maternal