ketonemia and hypoglycemia influenced fetal substrate levels as reflected in amniotic fluid obtained at termination of the fast and from 11 additional non-fasted PW. Ketone acid levels in amniotic fluid increased 30–40 fold in fasted PW to levels comparable to maternal blood (4–5 mM/L); glucose levels in amniotic fluid in fasted PW (21 ± 1 mg%) were 40% below those in non-fasted PW ($P < .001$). In contrast, free fatty acid levels in amniotic fluid were not consistently increased by starvation though markedly elevated in maternal plasma. Conclusions: Pregnancy accelerates and exaggerates the ketogenic and hypoglycemic response to starvation. Increased ketone availability to the conceptus suggests that ketones become an important metabolic fuel for the fetus during maternal caloric deprivation.

Hyperammonemia complicating parenteral nutrition in infants. J. JOHNSON, W. L. ALBRITTON, and P. SUNSHINE. *Stanford Univ., Stanford, Calif.*

The parenteral administration of protein hydrolysate-glucose mixtures is being used increasingly to prevent tissue catabolism and promote growth in infants with various conditions which preclude adequate ingestion or absorption of calories. Although nitrogen retention can be regularly attained, several complications have been reported.

We have found elevated blood ammonia levels in 5 of 6 infants receiving parenteral nutrition. In 4 of these infants, blood ammonia was >200μ g%. All 3 low-birth weight infants in the series developed hyperammonemia. Elevations of blood ammonia were seen during the infusion of either casein or fibrin hydrolysates. In 2 infants receiving long term infusions the level of blood ammonia correlated directly with the rate of infusion of protein hydrolysate whereas blood urea nitrogen levels did not change significantly. Elevations of serum transaminases and bilirubin accompanied hyperammonemia in 3 patients.

These data show that hyperammonemia is a common biochemical abnormality in newborn infants receiving parenteral nutrition with casein or fibrin hydrolysates at commonly employed infusion rates. Liver cell damage may accompany this mode of nutrition. The cause of hyperammonemia is unknown, but may be the result of an amino acid imbalance in the infusate.

The association of hypocalcemia with recurrent apnea of prematurity. JUAN J. GERSHANIK, ABNER H. LEVKOFF, and ROBERT DUNCAN. *Med. Univ. of South Carolina, Charleston, S. C.* (Intr. by Warren E. Wheeler).

Serum calcium, phosphorus, magnesium and total proteins were determined at 8 hour intervals during the first 48 hours of life in 27 neonates weighing under 1750 grams at birth, who were monitored for apnea. 14 babies developed recurrent apnea during the first 72 hours of life. The remaining 13 neonates had no distress. None of the 27 babies received calcium during the period of sampling. Calcium values were grouped into 9 consecutive 5 hour intervals and the mean value of the calcium levels in each interval was plotted using the mid-point of the 5 hour interval for time. Using the standard polynomial regression for a quadratic response, the apneic subjects showed a decrease in calcium values to a level of 5.9 mgm./100 ml. at 32 hours of age. The minimum mean calcium value for the non-apneic babies was 8.3 mgm./100 ml. at 32 hours of age. Apneic babies had higher phosphorus values and lower total serum proteins than the non-apneic babies. Recurrent apnea was associated with an increased maternal age and a higher incidence of previous abortion. Apneic babies had higher incidence of 1 min. Apgar below 5 (75% vs. 30%). Apneic spells developed in most of the cases during the first 24 hours of life (22.1 hours average). Thus, for the most part the onset of apnea precedes the development of hypocalcemia. Calcium urinary losses were similar in both groups. Calcium therapy appeared to reduce the number of apneic spells in 6 out of the 14 infants.

Low arterial oxygen tension: A primary event leading to periodic breathing and apnea in preterm infants. HENRIQUE RIGATTO, JUNE BRADY, FE DUMPIT, and WARREN TICKNOR. *Univ. of Calif., San Francisco, Calif.*

Nine babies (b.w. 1-2kg) were studied 38 times in the first 35 days of life. They were given 21, 19, 17 and 15% O_2 to breathe for 5 min each, then 21, 15 and 21% O_2 for 5 min each. We determined the incidence of periodic breathing, ventilation/apnea (V/A) time ratio, respiratory minute volume and frequency, heart rate, P_{AO_2} and P_{ACO_2}, and the P_{CO_2}, P_{C_2} and pH of arterialized blood. With 15% O_2 the incidence of periodic breathing was substantially increased (see Table); with 17% O_2 the incidence was less pronounced but significant ($P < .005$).

	Periodic breathing	V/A ratio	VE L/min/kg	Resp. Rate	Heart Rate	$PaCO_2$	$PACO_2$	PaO_2	PAO_2	pH
21% O_2	13%	2.0 ± .2*	.231 ± .017	36 ± 2	146 ± 2	42 ± 1	35 ± 1	68 ± 1	107 ± 2	7.319 ± .008
15% O_2	71%	1.4 ± .1	.192 ± .015	30 ± 2	151 ± 2	44 ± 1	35 ± 1	56 ± 1	69 ± 2	7.332 ± .008
P	<.005	<.005	<.025	<.005	<.005	<.001	>.05	<.001	<.001	>.05

Means ± SE.

* At onset of periodicity (baby breathing 21, 19 or 17% O_2).

In 3 babies the oscillations in oxygen saturation (ear oximeter) increased from 4% during 21% O_2 to 12% during 15% O_2. One baby became apneic (>20 sec) after prolonged periodic breathing with marked hypoventilation and low V/A ratio. These findings suggest that decreased P_{aO_2} may be a primary event leading to hypoventilation, periodic breathing and apnea in the preterm infant.

Visual estimation of body temperature in neonates. THOMAS K. OLIVER, JR. and ROBERT T. HALL. *Univ. of Pittsburg Sch. of Med., Pittsburgh, Pa., and Univ. of Missouri Sch. of Med., Columbia, Mo.*

Abdominal skin temperature of human neonates who are dressed and blanketed closely approximates core temperature. Although valuable in the detection of such illnesses as sepsis and hypoglycemia, the low yield of detecting abnormalities in term infants has resulted in temperature being measured at widely spaced intervals in most nurseries. This report describes a way of visually estimating body temperature using the cholesteric phenomenon. A mixture of cholesteric crystals was formulated

whose color was green between 36.3–37.1°C (normal abd. skin temp.). Below this temp. the color was brown; above, green-blue (37.2–37.9°C) or blue (>37.9°C). The crystals were fixed to black saran plastic with an adhesive undersurface which permitted fixation to the skin. 365 observations (65 infants) were made of the color of the abdominal tape compared to rectal temperature.

	Tape color				
	Brown	Green		Blue-green	Blue
# of observations	10	295		47	13
False positive (5.4%)	4	0		11	5
False negative (1.9%)	0	7	1 < 36.2°C 6 > 37.5°C	0	0

Conclusions: Body temperature of term infants can be estimated accurately, quickly, simply and frequently by this method.

The effect of phenobarbital on asphyxia of the newborn monkey. DAVID E. FISHER, JOHN B. PATON, HENRY H. MANGURTEN, and RICHARD E. BEHRMAN. *Univ. of Ill. Coll. of Med., Chicago, Ill.*

Fourteen newborn monkeys (*M. speciosa,* 2–6 days old) were asphyxiated; 8 were pretreated with phenobarbital and 6 were untreated controls. The treated group received 10 mg/kg I.M. twice prior to the asphyxiation; only one of the infants was not clinically sedated. After 5 minutes of total asphyxia (blocked endotracheal tube) there was a significant mixed respiratory and metabolic acidosis associated with a profound decrease in cardiac output (C.O. ml/min/kg microsphere withdrawal method). Regional blood flow (% C.O.) to the brainstem, midbrain, cerebellum (total of these 3 areas identified as Brain in table) and the heart increased; the % C.O. distributed to the G.I. tract and kidneys decreased. These effects of asphyxia were not significantly modified by phenobarbital.

Acid-base parameters, O_2 consumption ($\dot{V}O_2$-Kipp diaferometer), blood pressure, cardiac output, and the regional distribution of the cardiac output were determined for phenobarbital treated and control groups prior to axphyxiation; no significant differences were observed.

Mean values	pH	C.O.	Brain	Heart	G.I.	Kidney
Before						
Control	7.396	152	4.2	4.4	7.6	11.9
Phenobarbital	7.420	214	5.2	5.9	6.5	12.4
After						
Control	6.950	22	9.5	18.0	2.8	5.0
Phenobarbital	6.870	32	13.2	17.2	2.1	3.0

Red cell volume in respiratory distress syndrome. R. USHER, S. SAIGAL, A. O'NEILL, L. CHUA, and Y. SURAINDER. *Royal Victoria Hosp., Jewish General Hosp., and McGill Univ., Montreal, Que., Canada*

The role of placental transfusion in the development of RDS is still in dispute. Red cell volume is closely related to placental transfusion in prematures with 32 ± 3 ml/kg being found after immediate and 48 ± 6 ml/kg after 5 min delayed cord clamping.

In order to determine whether the degree of placental transfusion was related to the incidence or severity of RDS, red cell volume was measured at age 4 hours in 262 premature infants by I^{125} albumin and venous hematocrit × 0.87 to obtain total body hematocrit.

Red cell volumes averaged 39.7 ml/kg in unaffected premature infants and 37.1 ml/kg in those with RDS (PO.O2). RDS survivors had a higher red cell volume (37.7 ml/kg) than fatal cases (33.8 ml/kg). The 87 infants with the smallest red cell volumes (mean 30.1 ml/kg) had a 10.3% risk of death from RDS; the 88 with intermediate red cell volume (38.0 ml/kg) had a 5.7% risk, and the 87 with the largest red cell volumes (47.9 ml/kg) had only a 2.3% risk of death from RDS, even though the birthweights and gestational ages of the three groups were identical. From this data, a placental transfusion seems to greatly reduce the risk of death from respiratory distress syndrome.

Diagnosis of the respiratory distress syndrome (RDS) by the absence of phosphatidyldimethylethanolamine (PDME) in tracheal effluents of low birth weight (LBW) infants. PAUL Y. K. WU, ROBERT C. BORER, JR., and HOUCHANG MODANLOU (Intr. by Louis Gluck). *Los Angeles County-Univ. of Southern Calif. Med. Ctr., Los Angeles, and Univ. of Calif., San Diego, La Jolla, Calif.*

PDME, an intermediate in the synthesis of surface-active lecithin from lungs, was isolated from lipid extracts of tracheal effluent ("mucus") obtained from hypopharynx, separated by thin-layer chromatography on precoated silica gel mylar strips and detected with bromothymol blue.

PDME, arterial blood pH and rectal temperatures were recorded serially (½, 1, 2, 3, 4 hours of life) in 28 randomly selected LBW infants. Of 126 tests performed, the results at 3 hours correlated with diagnosis of RDS. PDME was absent in 14/16 infants with RDS, and PDME was present in 11/12 infants without RDS. There was a significant ($p < 0.01$) association between arterial blood pH and PDME:

pH 7.20 ± 0.10 S.D.	PDME absent
pH 7.27 ± 0.12 S.D.	PDME present

Little correlation was found between temperature and PDME. The results indicate that serial determinations of PDME may provide a rapid definitive early chemical diagnosis of RDS.

Early treatment of neonatal acidosis in low birth weight infants in relation to respiratory distress syndrome. C. J. HOBEL, M. A. HYVARINEN, A. ERENBERG, G. C. EMMANOUILIDES, and W. OH. *UCLA Sch. of Med., Harbor Gen. Hosp., Torrance, Calif.*

The effects of early vs late correction of acidosis on the clinical course of the respiratory distress syndrome (RDS) was evaluated in 82 pre-term infants weighing less than 2,250 grams. Criterion for inclusion into the study was a pH of less than 7.25 either from fetal scalp, cord or umbilical arterial blood samples within 20 min of age. The infants were randomly grouped into: A, birth weight <1500 gm, early treatment (intravascular $NaHCO_3$ infusion within 30 min of age); B, <1500 gm, late Rx (2–3 hrs of age); C, 1501–2250 gms, early Rx, and D, 1501–2250 gm, late Rx. Infants were otherwise similarly managed. At designated intervals predetermined clinical parameters were used to make the diagnosis and to grade the severity of RDS. No significant differences were observed in arterial blood pH and PO_2 values between groups during the first 30 min of life prior to Rx. In the early Rx groups, the arterial blood pH was significantly higher during the first 12