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