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kg/min) were administered to 7 infants with IRDS. In three patients the drug was infused directly into the pulmonary artery through an umbilical arterial catheter guided through the ductus arteriosus. A second umbilical catheter was placed in the aorta for pressure recording and blood sampling. In four other infants, the drug was infused into the superior vena cava.

In each of the 3 infants who received infusion into the pulmonary artery, a decrease in pulmonary artery pressure of 6 to 25 mm Hg was observed, accompanied by small increases in pH and PaO₂ and decrease in PaCO₂. The gradient established between aortic and pulmonary pressure persisted for periods up to 45 minutes but then disappeared while the infusion was still in progress. Clinical improvement was seen in only one of these infants and in none of the four who received infusion in the superior vena cava. It would therefore appear that acetylcholine is capable of promoting vasodilation of the pulmonary arterioles but is ineffective in treatment of IRDS. (APS)

89 Hypoglycemia in Infants with Erythroblastosis Fetalis.
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Hyperplasia of the Islets of Langerhans is usually found in fetuses in infants who die as a result of erythroblastosis fetalis (EF) and the pancreas contains levels of insulin comparable to those found in infants of diabetic mothers (IDM). The clinical counterpart of these observations, hypoglycemia, is believed to occur rarely if at all in infants with EF. During the last 18 months we have observed hypoglycemia (< 30 mg/100 ml in term infants; < 20 mg/100 ml pre-term) in 11 infants. Borderline levels have occurred in 2 others. Five of the infants developed hypoglycemia prior to exchange transfusion (ET). Recently 16 consecutive infants with moderate to severe EF have had frequent blood glucose measurements following ET. 6 (30 %) developed symptomatic hypoglycemia with borderline values in 2 others. 9 infants had multiple measurements of blood glucose in the 3 h period following ET and 4 of these had simultaneous measurements of serum immuno active insulin. Serum insulin was elevated in all 4 infants. Glucose disappearance constants (K_t) were calculated for the first hour following ET and compared to K_t values for normals and IDM following IV glucose tolerance tests. The infants with EF who developed hypoglycemia had significantly greater K_t values than did the normals; their values approximated IDM. Because of the necessity for prompt treatment, blood glucose should be measured frequently in infants with EF both after birth and after ET. Intravenous glucose or fructose should be effective in prevention of hypoglycemia. (APS)

90 Vascular Responses to Oxygen Breathing in the Newborn Infant. Nicholas M. Nelson*, Christopher H. Nourse*, Bettty L. Priestley*, Ruth B. Cherry* and Clement A. Smith, Department of Pediatrics, Harvard Medical School, Boston, Mass.

During the course of measurements of pulmonary function in newborn infants breathing 60–100 % oxygen, we have previously noted frequent, rapid and significant decreases in hematocrit and hemoglobin levels. The present investigation has been designed to

confirm and elucidate this finding. 14 newborn infants varying in gestational age from 36-37 weeks, in birth weight from 2.7-4.6 kg and in postnatal age from 1-18 h breathed 60 % O2 by demand valve for periods of 60-70 min. Hematocrit (Hct), hemoglobin (Hb), total protein (TP) and blood gases were followed throughout and blood volume (CO method) was measured in 3 infants. Within 10 min of onset of O₂ breathing prompt decreases of Hb, Hct, TP were seen reaching levels of 20 % below control values by 60 min. These changes promptly reversed upon resumption of air breathing. TP changes were seen to be more consistent and marked than changes in Hct or Hb. The response was seen with arterial O2 tensions of as low as 116 mm Hg. Total blood volume increases of 40-60 ml/kg were noted during O2 breathing in 3 infants. These data suggest auto-infusion of tissue fluid from some vascular bed in response to O₂ breathing; this may imply an increase in precapillary resistance with consequent decrease in capillary pressure and derangement of the Starling equilibrium. Preliminary investigations indicate that bradycardia and increased peripheral vascular resistance are involved in this phenomenon. (APS)

91 Otic Lesions and Congenital Hypothyroidism. Gerald J. Bargman* and Lytt I. Gardner, State University of New York, Upstate Medical Center Syracuse, New York.

In an effort to elucidate the relation, if any, between thyroid abnormality and congenital deafness in Pendred's syndrome, an experiment was designed to study the effects of hypothyroidism on middle and inner ear hearing structures, including the auditory nerve and its central projection, in developing chick embryos. Propylthiouracil (PTU), 2 mg, was injected into the albumin of fertile chick eggs on the 10th incubation day. Single doses of 1-thyroxine (range 1 to 100 μ g) were inoculated in a similar manner, either alone or with PTU. Control inocula included sterile saline or water. After hatching, each chick was examined for obvious malformations. The thyroid glands, middle and inner ear mechanisms, auditory nerve and brainstem were studied grossly and with different histologic staining techniques. When compared to controls, chicks exposed to PTU on their 10th incubation day exhibited: increased mortality, delayed hatching, reduced size, incomplete yolk sac absorption and death within 5 days unless exogenous thyroid hormone was provided in the first 24 to 47 h post hatch. Specific, consistent, morphologic alterations were observed in their thyroid glands as well as in the sensory hair cells and spiral ganglion of the cochlea. Preliminary results indicate if $50-75~\mu g$ of 1-thyroxine is given simultaneous with (or as long as 120 h after) the PTU injection of the 10 th incubation day, one cannot detect the groos defects, marked thyroid lesions or abnormal histology in cells of the cochlea and its ganglion. A relationship between embryonic thyroid gland function and the hearing mechanism of the chick embryo, is suggested. (SPR)

92 Mechanism of Myelin Formation During Development. NORBERT HERSCHKOWITZ* and GUY M. McKhann, Stanford Univ. Sch. of Med., Palo Alto, Cal.

Myelin is formed by a satellite cell which wraps around the axon to form a continuous spiral of membrane. This membrane has a distinctive lipid and protein composition when compared with other membran-