hormone deficiency in 6 with multiple tropic hormone deficiencies in 4/6. In 3 of 4 patients, in whom a PEG was performed, absence of septum pellucidum, thin optic nerves and chiasm, abnormal fornices, irregular lamina terminalis, and an abnormally shaped 3rd ventricle with small inferior pointing diverticulum from the optic recess were demonstrable. The sella turcica and suprasellar cistern were normal. EEG dysrhythmias were noted in 2 of 4 patients tested; I.Q. was normal or mildly subnormal in 6. No evidence of ocular abnormalities or dwarfism was noted in the parents or sibs of the 6 patients. Hence, sporadic hypopituitary dwarfism is not uncommonly associated with a characteristic congenital malformation of the prosencephalon. The hypopituitarism is ascribable to a diencephalic defect which results in deficiency of hypothalamic hypophysiotropic factors and in some cases vasopressin.

177 *Two Types of Congenital Cysts of the Posterior Fossa: A Comparison of Their Clinical and Pathological Characteristics and Their Embryogenesis.* JEROME S. HALLER, SAMUEL M. WOLPERT and EDWARD F. RABE, Tufts Univ. Sch. of Med., New England Med. Center Hosps., Depts. of Ped., Neurol., and Radiol., Boston, Mass.

Two types of cystic lesions can occur in the posterior fossa of infants and children. In one type there is a cystic dilatation of the 4th ventricle known as the Dandy-Walker (D-W) syndrome, and in the second type, a cyst or cysts overlie the cerebellum. This is referred to as a posterior fossa extra-axial cyst (PFEAC). These two entities may be differentiated clinically as will be illustrated by presentation of the findings in 2 infants with PFEAC and 2 with D-W syndrome. The pattern of skull transillumination is distinctive for each entity, although the differences are subtle, and can enable the clinician to suspect the correct diagnosis immediately. Arteriographic and air contrast findings show similar abnormalities in the supratentorial region in both conditions but distinctive differences are present in the infratentorial region. The histology of the cyst wall in PFEAC differs from that in D-W syndrome. Inferences drawn from a comparison of the arteriographic and histologic findings in these 4 cases with the appearance of certain brain structures at different stages of human embryogenesis indicate that both conditions are congenital abnormalities occurring before the 3rd fetal month but affecting the leptomeninges in one instance (PFEAC) and the cerebellar anlage in the other (D-W syndrome).

178 *Quantitative RISA Subarachnoid to Plasma Transport and Subarachnoid Perfusion Tests in Children with Progressive Macrocephaly and Suspected Hydrocephalus.* THOMAS H. ROCKEL and EDWARD F. RABE, Tufts Univ. Sch. of Med., New England Med. Center Hosps., Dept. of Ped. and Neurol., Boston, Mass.

A measure of the adequacy of lumbar subarachnoid to plasma CSF transport is obtained by determining quantitatively the percent of ^{12}/I RISA in the plasma 24 h after its lumbar subarachnoid injection. This was done in 35 infants and children ranging in age from 5 weeks to 14 years. The ability of the CSF circulation to dispose of normal saline perfused into the lumbar subarachnoid space at a rate of 0.76 ml/min for 30–60 min was determined by CSF pressure measurements at 5-min intervals during the perfusion of a sedated patient. This was done also on 17 of the 35 patients. Patients with normal external CSF circulation transported 38.5±10.2% (2 S.D.) of the injected RISA; patients with communicating hydrocephalus, 16.1–25.4%; those with obstructive hydrocephalus, 31.5–45.8%; and macrocephalic achondroplastic dwarfs, 29.9–53.1%. The pattern of perfusion test CSF pressures could be divided into 3 distinct forms: normal, uncompensated abnormal and partially compensated abnormal. In every instance save one, the results of the two tests on the same patient agreed. This patient with hydrocephalus and porencephaly had a normal RISA transport test but an abnormal uncompensated perfusion pattern. From these data we conclude, (1) that both tests are needed to evaluate the adequacy of the external CSF circulation, and (2) the external circulation can be normal when the internal circulation (ventricles to cisterna magna) is obstructed.

179 *Failure of Exchange Transfusion to Prevent Minimal Cerebral Damage When Employed so as to Maintain Serum Bilirubin Concentrations Below 18 and 20 mg/100 ml.* LOIS H. JOHNSON and THOMAS R. BOGGS, Dept. of Ped., Pennsylvania Hosp., Philadelphia, PA.

The occurrence of minor cerebral deficit in the absence of gross neurologic damage as a result of neonatal hyperbilirubinemia has been suspected for some time [DAY and HAINES, Pediatrics *00:* 000, 1954]. Recently ODELL (J. Pediat. *00:* 000, 1970] has documented the presence of such damage by means of psychometric and neurologic evaluations at age 5 to 6 years in a group of jaundiced infants treated with exchange transfusion. Damage was found to correlate with degree of saturation of the serum proteins with bilirubin (as measured by his salicylate saturation index). We are confirming these results in a similar study using our HABA technique (Program SPR 1966, COLEMAN, Jr. 50 L serum, cc 30×10^{-5} HABA, PO_4 buffer) as a measure of serum binding reserve.

Analysis of the first group of infants being recalled at age 4 years for detailed psychometric, speech and hearing and neurologic examinations suggests that jaundiced infants whose HABA binding levels fall below 50% are likely to suffer some degree of measurable damage which infants whose binding levels remain above this level will escape (Chi square = 6.04, $p = 0.014$, $n = 41$). Correlation of damage with bilirubin/albumin molar ratio was good. As in the study of ODELL, correlation with serum bilirubin concentration was poor.

The data clearly indicate that if minimal as well as major cerebral damage as a consequence of neonatal jaundice is to be prevented more exchange transfusions will have to be done or some alternative or additional form of therapy will have to be used. Phototherapy in conjunction with exchange transfusion gives promise of providing increased protection without increased therapeutic risk. Its ability to do so should be subjected to the scrutiny of long-term follow up studies.

180 *Effects of Hyperoxia on the Nucleic Acid Contents of Developing Brain.* GILMAN D. GRAVE, CHARLES KENNEDY and LOUIS SOKOLOFF, Lab. of Cerebral Metabolism, Nat. Inst. Mental Health, Bethesda, MD.

Prolonged exposure of the newborn to elevated concentrations of oxygen may initiate pathologic changes in the retina, but, except for inhibition of capillary