ous structures. The myelin components may be synthesized at the level of the forming membrane, or transported into the membrane following synthesis by some other intracellular organelle.

The synthesis of sulfatide, a membrane lipid, by 9[5] and 17-day-old rats was determined after a single intraperitoneal injection of ^{35}S-sulfate into the sulfatide of whole brain, myelin, non-myelin organelles, and 105,000 × g supernatant of brain was followed. At times when there was no further *de novo* synthesis of lipid, the amount of labelled sulfatide in myelin continued to increase while that of non-myelin membranes decreased. The transfer of sulfatide from non-myelin structures to myelin appeared to be mediated by soluble lipoproteins which were isolated from supernatant, characterized, and found to contain labelled sulfatide.

In vitro studies, indicated that isolated myelin contained galactocerebroside sulfokinase, the enzyme involved in the synthesis of sulfatides. Thus, isolated myelin contains some membranous components capable of synthesizing myelin lipids.

These studies suggest that a lipid component of myelin is both transported into the forming membrane by a carrier lipprotein and synthesized by the myelin membrane. The relationship between these two mechanisms of formation of myelin components during development will be discussed. (SPR)

93 *Physico-chemical Properties of Ceruloplasmin from Normal and Wilson's Disease Subjects.* NEIL A. HOLTZMAN* and MICHAEL A. NAUGHTON*, Johns Hopkins Univ. Sch. of Med., Baltimore, Md. (introduced by Robert E. Cooke).

Although most patients with Wilson's disease have low levels of serum ceruloplasmin, an occasional patient presents with a normal level. This variation might be due to differences in the structure of ceruloplasmin. To investigate this possibility, physico-chemical properties of ceruloplasmin from a Wilson's disease patient with normal levels of ceruloplasmin, from a second, unrelated, patient with low levels and from normal subjects were compared.

Highly purified ceruloplasmin from the patient with normal levels was chromatographed on hydroxylapatite. The major component obtained had the same electrophoretic mobility (at pH 5.6, 6.4, 8.9), tryptic peptide map, oxidase activity, copper, amino acid and sugar composition as the major component from normal subjects. The minor hydroxylapatite component from the patient had the same electrophoretic mobility (at pH 5.6), tryptic peptide map and oxidase activity as the minor component from normal subjects. The major and minor ceruloplasmin components were found to differ from each other by only one peptide spot. Ceruloplasmin from the Wilson's disease patient with a low serum ceruloplasmin concentration had the same chromatographic and electrophoretic characteristics as normal ceruloplasmin. Thus it appears that ceruloplasmin in Wilson's disease is structurally normal.

Penicillamine administered to the patient with normal ceruloplasmin concentration caused an increased excretion of copper. Concommitantly, ceruloplasmin activity became undetectable in his plasma and reappeared only after the drug was discontinued. This finding suggests that in Wilson's disease, the sensitivity of the ceruloplasmin synthesizing system to copper may be diminished. (SPR)

94 *Effect of Hypertonicity on the Metabolism of Brain in Vitro.* JEAN F. SOTOS, S. ADDANKI*, P.D. REARICK* and F. D. CAHILL*, Depts. Ped. & Physiol. Chem., Ohio State Univ. Col. of Med. & Children's Hosp. Res. Fndn., Columbus, Ohio

Hypertonicity of body fluids whatever the cause—hypernatremia, uremia, hyperglycemia—alters CNS function with alteration of sensorium, coma and death. Also, hypdtonicity induces acidosis (SOTOS *et al.:* Pediatrics *26:* 925; *30:* 180). The mechanism of the alterations is unclear. Rat brain slices where incubated in Krebs-Ringer phosphate buffer containing glucose-UL-C-14 or pyruvic acid-2-C-14 as substrates in 100 % O_2 atmosphere (ADDANKI *et al.:* Ann. Biochem. *13:* 458 [1965]). Using glucose as substrate, increases in tonicity due to NaCl, sucrose or glucose (340 to 1000 mOsm/kg H_2O) caused decreases in O_2 consumption (97 to 100 %). The same hypertonic media did not change O_2 consumption of rat brain homogenate. Urea up to 1000 mOsm had no effect on O_2 uptake of slices. The $C^{14}O_2$ evolved decreased to 80 and 10 % of control with hypertonic media containing 450 and 900 mOsm of either NaCl or sucrose with glucose as substrate. These changes were due to decreased Krebs cycle activity, since similar values were obtained when pyruvic acid-2-C-14 was used as substrate. No effect was seen with urea solutions at 450 mOsm, decreasing to 81 % of control at 900 mOsm. Studies conducted with glucose 1-C-14 or 6-C-14 did not show any significant change on the activity of the hexomonophosphate shunt. The different values can be accounted for by the different osmotic effect of the solutions. The lactic acid production increased 160 % of the control with NaCl hypertonic media. These observations bear on the pathogenesis of (acidosis and) CNS alterations observed in clinical conditions with hypernatremia, hyperosmolar non-ketotic diabetic coma and experimental uremia. This investigation was supported by USPHS grants NB 05114 and FR-78. (SPR)

95 *Continuous Recording of Cerebral Ventricular Fluid Pressure in Hydrocephalic Children.* PATRICIA HAYDEN*, ELDON FOLTZ and* DAVID B. SHURTLEFF, Univ. of Wash. Med. Sch., Seattle, Wash. (introduced by Ralph Wedgwood).

To quantitate physiologic variations in and the effect of chemical agents on cerebral intraventricular pressure a technique for continuous pressure recording via an indwelling ventricular catheter and strain gauge transducer has been developed. Pressure studies for periods up to 10 days have been made on 12 hydrocephalic children ranging in age from 3 weeks to 5 years. Baseline recordings extending over 24 to 96-hour periods revealed multiple patterns of rhythmic variation with mean pressures ranging from 280 to 50 mm of water. Other physiologic parameters studied included respiratory rate and pattern, electrocardiogram and scalp venous pressure. Serum and ventricular fluid electrolyte and osmolar concentrations have been monitored. Ten of 11 children given oral isosorbide showed an immediate decrease in ventricular pressure ranging from 53 to 100 % of baseline similar to the effect of mannitol and urea. Ventricular wave form flattened during periods of pressure reduction. Five patients studied were also given acetazolamide, urea and mannitol. Acetazolamide increased ventricular pressure 2 to 3 fold with a concommitant exaggeration of pressure wave form. This hypertensive effect was immediate and persisted 2 to 4 hours before returning to