intestinal transport. J. F. Desjeux and C. L. Morin. *Hôpital Sainte Justine, Montreal, P.Q., Canada.*

The purpose of this study is to know if saliva and serum of cystic fibrosis have a general activity on membrane transports and if there is a common abnormal factor in blood and saliva. The method measures the variations of rat intestinal uptake on alanine which is in relation with Na movements. Two groups of 10 children with cystic fibrosis were compared with a pool of normal children. The fresh saliva of the first group was in contact for 10 min with an everted ring of normal rat jejunum. The ring was then incubated 60 min in K.R.B. with 0.8 mm/liter ^{14}C-alanine. The fresh serum of the second group was placed in the incubation medium for 60 min with ^{14}C-alanine. The saliva of cystic fibrosis inhibits alanine uptake from 6 to 50% compared with normal pool saliva. These results seem constant with a dilution from ¼ to 1/20. No significant difference could be obtained with fresh or congealed serum with a dilution from ¼ to 1/20. Saliva (and, perhaps, other exocrine secretions) of cystic fibrosis has a general effect on cellular uptake (probably on Na uptake). But it seems that this factor is different from the abnormal factor of the serum of cystic fibrosis.

11. Fatal congenital lactic acidosis in two siblings. S. O. Lie, S. Skrede, and J. H. Strømme. *Univ. of Oslo, Norway.*

Chronic lactic acidosis has been described in children. We have studied two siblings who developed a severe metabolic acidosis during the 2nd day of life. Lactic acid levels of 88–108 mg 100 ml blood were found. The children were severely affected, and the disease showed a malignant course with death at the ages of 97 and 177 hr.

In vivo as well as *in vitro* studies were carried out in the second sibling in an attempt to localize the metabolic defect. Metabolism of lactate was determined by injecting lactate-2-^{14}C intravenously. Postmortem examinations included determinations of liver and muscle glycogen, LDH-isoenzymes, the metabolism of radioactive lactate and pyruvate in liver and muscle slices and in isolated mitochondria. The studies indicated that the hyperlactatemia was due to an abnormally slow metabolism of lactate and not to a high production. The rate of oxidation was normal. Very low concentration of glycogen was found in liver and muscle. The incorporation of ^{14}C-lactate into blood glucose and glycogen was insignificant *in vitro* as well as *in vivo*. These results are compatible with a reduced gluconeogenesis from lactate in our patient, resulting in a severe accumulation of lactate and a fatal acidosis.

12. Studies on thyroid proteins in congenital goiter. P. Olin. *St Göran's Hosp. and Karolinska Hosp., Stockholm, Sweden.*

Congenital goitrous hypothyroidism may be caused by a metabolic bloc in the synthesis of the thyroid hormones or by an abnormal synthesis of iodinated proteins in the thyroid. Previously we have utilized labeling *in vitro* with ^{3}H-leucine and Na^{125}I in the study of the normal synthesis of 17-19 S thyroglobulin in the human fetal thyroid gland (Olin, Vecchio, Ekholm, and Almqvist: Endocrinology, *86:* 1041, 1970). This system has been applied to 2–10 mg specimens from congenital goiters obtained by percutaneous biopsy.

This report deals with two children with a pathological perchlorate-induced discharge of radioiodine. The first patient was an 18-day-old boy with diffuse goiter and hypothyroidism. The biochemical analysis revealed a normal uptake *in vitro* of ^{125}I. No ^{125}I was present in the crude extract after dialysis. Sucrose density gradient centrifugation of the soluble proteins, however, showed a distinct label of ^{3}H in the 17-19 S region. The ^{3}H-radioactivity was specifically precipitated by antiserum against human adult thyroglobulin.

The second patient was a 4-year-old girl with goiter and hypothyroidism since at least 1 year of age. At that time the perchlorate test was performed before treatment. The biochemical studies were performed while the patient was treated with thyroxine. Biopsies were obtained before and after stimulation with TSH (Actyron 1 IU/d for 5 days). Before TSH stimulation no iodide was accumulated *in vitro* and a slight ^{3}H-peak was present in the 17-19 S region of the sucrose density gradient. After TSH stimulation the thyroid tissue did accumulate ^{125}I, but only ^{3}H was incorporated into the 17-19 S proteins.

The results indicate a normal synthesis of the protein moiety of thyroglobulin in these two congenital goiters with a defect in the organification of iodine. This is at variance with the current opinion that this type of goiter is caused by a deficient synthesis of thyroglobulin and a pathological formation of thyralbumin.

13. Early postnatal weight gain of low weight newborns: Relationships with various diets and with intrauterine growth. E. Rezza, U. Colombo, G. Bucci, M. Mendicini, and S. Ungari. *Univ. of Rome, Italy.*

From 10 to 34 days of age, the weight gain was studied in 134 low birth weight (LBW) infants (birth weight range 0.99–1.90 kg; gestational age range 28–38 weeks) on three different diets: (*1*) human milk; (*2*) a "humanized" milk formula; (*3*) a high protein, high CI, low fat cow's milk formula. The caloric intake (kcal/kg/24 hr) was always about 120 between 10 and 20 days, and about 140 thereafter. Six experimental groups were separated according to the three diets, and whether infants were "large for date" (LFD, *i.e.*, with birth weight > the 25th percentile for gestational age) or "small for date" (SFD, *i.e.*, with birth weight ⩽ the 25th percentile, and 50% of subjects ⩽ the 10th percentile). Weight gain curves were calculated by regression analysis using the least square method according to a polynomial model. In each group, an exponential relationship of weight gain with time was found. In SFD infants weight gain was slightly but significantly ($P < 0.001$) faster than in LFD babies on *diet 3*, not on *diets 1* and *2*. Either in LFD or in SFD infants weight gain was markedly faster on *diet 3* as compared to *diet 1* or *2* ($P < 0.001$), and slightly but significantly ($P < 0.01$) faster on *diet 1* as compared to *diet 2*. Previous findings of a faster weight gain of LBW newborns on high protein formulas, as compared to human or "humanized" milk formulas, were therefore confirmed. The following main conclusions and speculations were also made: in studies on growth refeeding, LFD and SFD newborns should be discriminated, and statistical methods able to describe a possible nonlinearity of growth curves should be used; SFD babies presumably need more proteins than LFD infants for optimal weight gain in the early weeks of life.

14. Changes in the lipid pattern of human sera in the neonatal period. J. Clausen and B. Friis-Hansen. *Rigshospitalet, Copenhagen, Denmark.*

The changes in serum β-lipoprotein and the relative distribution of lipoproteins as well as the pattern of fatty acids have been followed in infants during the neonatal period. The data obtained were correlated to the food intake as well as to the growth of the child. In infants living on human milk, the total content of β-lipoprotein increased from around 30% of the concentration in the mothers' serum at birth, to around 80% in the 2nd week of life. During the same period the lipoprotein pattern changed from a predominance of the pre-β-lipoprotein band to a pattern