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Changes of erythrocyte membrane fatty acids and systems of their protection against peroxydation during the first year fo life.

Vitamin E is supposed to protect erythrocyte membrane fatty acids against peroxydation and prevent hemolytic anemia. But hemolysis is not a necessary consequence of vitamin E deficiency; therefore the natural course of other variables concerning lipid peroxydation was studied during the first year. Serum vitamin E,glutathione peroxydase activity(gl.p.a.),erythrocyte membrane fatty acid momposition were determined at birth(n=23),1(n=6),2(n=13),4(n=7),6(n=9) and 12(n=7)months of age. In addition erythrocyte selenium concentrations were measured because of the dependency of gl.p. on this trace element. Serum vitamin E levels were 0.45±0.25mg/dl at birth compared to 0.94±0.16mg/dl at 12 months(p 0.01).Erythrocyte gl.p.a. at birth(33.0±8.1 IU/$10^8$cells)compared to the activity at 12 months(28.8±1.8 IU/$10^8$cells)was not significantly different.Erythrocyte selenium concentrations were higher at birth(2.35±0.75 µg/$10^8$cells)compared to 12 months(1.21±0.55 µg/$10^8$cells)(p 0.01).Erythrocyte membrane fatty acids were analysed from 14:0 to 26:1. Among the individual fatty acids there was a distinct increase of 20:4 and 24:0 and a decrease of 22:6 concentrations. Multiple correlations were performed.

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RELATION BETWEEN PLASMA SULFATE ($SO_4$), PHOSPHATE ($P_i$) AND RENAL FUNCTION IN CHILDREN WITH CHRONIC RENAL FAILURE (CRF).

Hypersulfataemia is a common finding in advanced CRF. In this study we determined $SO_4$ in various stages of CRF in 20 non-dialysed children age 4-18 yrs. in relation to GFR, ERPF, other plasma electrolytes and acid base balance. $SO_4$ was measured by precipitation with $BaCl_2$ and subsequent atomic absorption spectrophotometry of Ba in the supernatant.GFR ($C_{Inulin}$) ranged between 4 and 66 (mean 28) ml/min/1.73 $m^2$ BSA and ERPF ($C_{PAH}$) between 13 and 305 (mean 125) ml/min/1.73 $m^2$. In all cases $SO_4$ was > 0.3 mM/l(upper limit of normal) at GFR < 45 ml/min/1.73 $m^2$. $SO_4$ raised exponentially with decreasing renal function to levels 10 times normal. Significant correlations were found between $SO_4$ and $C_{Inulin}$ (r = 0.92), $C_{PAH}$ (r = 0.87), serum creatinine (r = 0.74), BUN (r = 0.63) and inorganic phosphate (r = 0.73), respectively, whereas serum Ca and base excess failed to correlate with $SO_4$. In conclusion, the increase of plasma $SO_4$ seems to depend primarily on renal function and less on the nutritional state of the uremic child as expressed by BUN. The close correlation between $P_i$ and $SO_4$ may be an indicator for a possible role of sulfate metabolism in the pathogenesis of renal osteodystrophy.

**81** W. Lukas, J. Lewandowski, S. Halvorsen/Intr. by S. Halvorsen/ I Clinic of Pediatrics Zabrze Pol. Ulleval Hospital Oslo. Norway. NEW SCREENING METHOD FOR HYPOTHYROIDISM.

Recent development of neonatal screening for hypothyroidism with the use $T_4$ measurement in small quantities of serum indicates that in premature babies $T_4$ level frequently lower than 2 S.D. below the mean value is due to RDS or small body weight. To reduce the number of false-positives in $T_4$ screening program for neonatal hypothyroidism low cost screening method of distribution binding ratio of thyroxine/DBR/with TBG and TBPA was prepared. The method is based on electrophoresis of serum sample obtained from U tubes on agarose plates. For direct DBR determination as well indirect determination of endogenous level of $T_4$ and TBG capacity 2 testing doses of radioactive $T_4$ $I^{125}$/ lug/dl/together with non-radioactive $T_4$/15,30 ug/dl/were added to the 20 ul of examined serum. 200 neonates and 50 premature babies/20 with small body weight below 1700 g and 4 with RDS/ were screened. Besides influence of physical factors on DBR were tested. No essential influence of physical factors on DBR value was proved. However correlation among pH, $pO_2$ of blood and DBR was noticed. In prematures babies with RDS and small body weight DBR values and TBG capacity were lower than in full term newborns.

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Sudden infant death syndrome and changes in the endocrine pancreas.

Lymphocytic infiltration in the pancreas of a victim of sudden infant death syndrome (SIDS) led us to investigate closer the possible changes in the endocrine pancreas. We have analysed 35 consecutive cases, aged 1-10 months. The infants had usually had a mild prodromal respiratory infection. The pancreas specimens were stained with hematoxylin-eosin and thiosulfation aldehyde-fuchsin. Diffuse islet hyperplasia and/or nesidioblastosis-type islet proliferation were found in most cases, and in some cases infiltrative islet tissue growth. The proportion of the islets of the whole parenchyma was larger in SIDS than in children who died from known causes (5.79 ± 0.31 % vs. 3.64 ± 0.47 %, mean ± SEM, P< 0.001). Serum insulin was measured by radioimmunoassay. Serum IRI values are available from four SIDS, the values being < 1 µU/ml in three cases and 6 µU/ml in one case, as compared to 6,7,10 and 11 µU/ml in four controls. Our assumption is that severe hypoglycemia might explain the death in some cases of SIDS. The possibility of abnormal circulating insulin forms should be explored. A viral infection might trigger the fatal sequence of events.

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Familial nesidioblastosis.

Nesidioblastosis indicates an abnormal formation of islets and endocrine cells from the pancreatic duct epithelium. The condition usually manifests itself as hypoglycemia of early infancy. We here present a female infant (birth weight 6190 g), who was the fifth child of a non-diabetic mother. Babies Nos. 1 and 2 had birth weights of 5700 and 5320 g, respectively, and both died neonatally. In baby No. 2 islet cell hyperplasia was noted at autopsy. Children Nos. 3 and 4 had normal birth weights, and are living healthy. In the present case hypoglycemia could not be managed with diazoxide, and a subtotal pancreatectomy (5/6) was performed at the age of 2 months. Specimens were taken for histological, immunocytochemical, ultrastructural, and radioimmunological investigation Preliminary results indicate a nesidioblastosis with amply occurring insulin-producing cells, but without any islet-cell tumors (or "microadenomas"). Operation and subsequent treatment with zinc-protamine glucagon gave only transient improvement. Somatostatin (10 µg/kg/hr i.v.) was effective in raising the blood glucose, but longacting somatostatin was not available. Shortly before a second operation was scheduled, the patient died suddenly. Autopsy revealed a marked regeneration of the remaining pancreas.

**84** L.MADÁCSY [X]/Intr. by H.K.Åkerblom/.Children's Hospital,Miskolc,Hungary. Kidney size and renal function in poorly and well-controlled diabetic children.

It has been shown earlier that glomerular filtration rate /GFR/ and kidney size is increased in early childhood diabetes. The purpose of the present study was to investigate whether a connection could be demonstrated between the kidney size and the metabolic control of the disease. Renal function and roentgenographic kidney size were measured in 8 children with newly diagnosed diabetes, in 22 insulin treated diabetic children with a short-term duration of the disease and in 12 matched controls aged 7-14 years. Kidney size and GFR were significantly increased in newly diagnosed diabetics and in 12 poorly controlled diabetic children, but there was no significant change of renal size or GFR in 10 well-controlled diabetic children compared to the controls.The results suggest that there is a basic connection between the kidney size respectively the renal function and the metabolic control in early childhood diabetes.