29 Cerebroprotective effect of dexamethasone (DXM) in newborn piglets with experimental pneumothorax (EPT). P. TEMESVARI[x], F. JOO[x], M.KOLTAI[x], E.BOK[x], G.ADAM[x] and D.BODA. Pediatric Dept., Univ. Med. School, Szeged, Hungary. The effect of DXM treatment in newborn piglets with EPT was studied on 30 piglets. 20 animals were subjected to EPT (group A and B) and 10 piglets served as controls without EPT (group C). In the EPT animals 10 piglets were injected with 5 mg/bwkg sc DXM 4 hours prior to EPT (A). Each animal was paired with untreated sibling having the same EPT (B). In the EPT piglets 4 hours after the critical phase (as apnea appeared, MABP fell, the EPT was terminated) brain tissue water and Evans blue dye were determined. There was a highly significant difference between A and B piglets regarding the time necessary to the development of critical phase (A X=69.6±SD=27.5 vs B 28.7±10.1 minutes; $p < 0.001$). In the untreated EPT animals (B) the Evans blue content of the parietal cortex and cerebellum exceeded significantly the values obtained in DXM treated (A) and control piglets (C) - parietal cortex A 0.18±0.12, B 0.88±0.57, C 0.17±0.07; cerebellum A0.14±0.02, B 0.78±0.36, C 0.17±0.07 µg/dye/g wet tissue. The same phenomenon was observed regarding the water content of above brain regions, as well. In conclusion, DXM treatment increases the tolerance to hypoxia and prevents brain oedema in newborn piglets with EPT.

30 T-CELL DEFICIENCY IN AN AFRICAN FAMILY. T. Jonckheer[1], P. Van de Perre[1], P. Lepage[2], H. Taelman[3], C. Muller[1], N. Clumeck[1], I. Dab[1]. 1. Depts Ped., Inf. Dis and Path. St. Peter Univ. Hosp, Brussels. 2. Hôpital Kigali Rwanda. 3. Tropical Institute Antwerp.

We report the case of a 19-month old African boy born from parents with clinical and immunological features suggesting prodromal acquired immunodeficiency syndrome (AIDS). He showed early failure to thrive, generalized lymphadenopathy, bilateral parotitis. Later he developped persistent interstitial pneumonia, persistent oral thrush and mild hepatosplenomegaly. Immunological studies showed hyper IgG level (3.2 g/dl), reversed T-helper/T-suppressor (T4/T8) ratio (19/68) and T-cell defect (cutaneous anergy for different antigens, depressed in vitro response for concanavalin, phytohaemagglutinin and Pokeweed mitogens). Open lung biopsy showed interstitial nodular lymphocytic infiltrates on optic microscopy and cytoplasmic particles on electron microscopy which are of uncertain origin. Recently the two older brothers were investigated. The oldest (6 years) showed chronic parotitis, hyper IgG level (4.7 g/dl), reversed T4/T8 ratio (18/49) and depressed in vitro response to mitogens. The second (5 years) showed slight parotitis, reversed T4/T8 ratio (29/32) and depressed in vitro response to mitogens. Both had normal chest X-rays The clinical and immunological presentation of the three children resemble that of infants with AIDS-like syndrome described by others in the USA. We suggest specific modes of transmission could be involved in familial acquiring of AIDS (transplacental-route, breast feeding, or other routine close-contact).

31 INFLUENCE OF GROWTH HORMONE (GH) TREATMENT ON INSULIN SENSITIVITY AND INSULIN RECEPTOR BINDING IN PATIENTS WITH GH-DEFICIENCY. H.Frisch[+], R.Prager[+], E.Schober[+], G.Schernthaner[+] (Introd. by W.SWOBODA). Pediatric Dept., Dept. of Medicine II, Univ.Vienna and L.Boltzmann Institute for Endocrinology, Vienna, Austria.

In patients with GH-deficiency various disorders of carbohydrate metabolism have been reported. The aim of the study was to examine the influence of GH on insulin sensitivity and insulin receptor binding by means of euglycemic CLAMP technique and radioreceptor assay using mononuclear leucocytes as target cells. 5 patients with hypopituitarism were studied during GH therapy (phase A) and after withdrawal of treatment for 4 weeks (phase B). Informed consent from the patients and their parents to partizipate in the study was obtained. Results: 1.) Basal insulin levels were not different in phase A and B. 2.) Peripheral glucose utilisation was significantly elevated in phase B compared to phase A (6.3 vs. 3.9mg/kg BW·min, $p < 0.05$). 3.) Insulin receptor binding to monocytes remained unchanged after withdrawal of GH-therapy (specific binding fraction 3.18% vs. 3.66%, n.s.). These data indicate that the influence of GH substitution on peripheral glucose metabolism is most likely due to postreceptor mechanisms.

32 Surfactant and sudden infant death (SIDS) MORLEY CJ, HILL CM*, BROWN BD*, BARSON AJ*, SOUTHALL D*, DAVIS JA. Dept of Paediatrics Addenbrooke's Hospital, Cambridge. Dept Pathology St Marys Hospital, Manchester, Cardiothoracic Unit, Brompton Hospital, London.

To detect abnormalities of pulmonary surfactant in SIDS surfactant was lavaged at autopsy from the lungs of 59 infants who died from SIDS, analysed for its phospholipid composition and compared with the composition of surfactant from 39 babies dying from HMD, 8 newborn with normal lungs, 9 infants with normal lungs, 8 tracheal aspirates from living adults and 15 from living infants. The percentage phosphatidylcholine (PC) was reduced in SIDS and sphingomyelin(SM) increased as shown. mean±sem.

		%PC (p)			%SM (p)	
SIDS	59	60 ± 1		12 ± 1		
HMD	39	63 ± 2	ns	9 ± 1	p < .05	
Newborn	8	78 ± 2	p < .01	2 ± 1	p < .01	
Dead infants	9	70 ± 2	p < .01	6 ± 1	p < .01	
Live infants	15	70 ± 3	p < .01	3 ± 1	p < .01	
Live adults	8	76 ± 4	p < .01	1 ± 1	p < .01	

For the individual groups there is some overlap of these parameters. However the ratio of PC:SM is a reasonable differentiator. The 90th centile PC:SM ratio for SIDS is 10. At this level there are 22% newborn, 22% of dead infants, 20% live infants, 13% live adults and 67% HMD. In conclusion the changes in surfactant composition whether pathological or developmental, is such a constant feature of SIDS that it is likely to be a fundamental component of the mechanism that contributes to the sudden death.

33 NUTRITIONAL IRON DEFICIENCY (NID) DELAYS THE MATURATION OF INTESTINAL ABSORPTIVE FUNCTIONS BY ALTERATION OF SURFACE MEMBRANE GLYCOPROTEIN SYNTHESIS. J.P. Buts, D. Delacroix*, F. De Craeker*, N. De Keyser* and R. De Meyer*. Dept. of Ped., Univ. of Louvain, Brussels, Belgium.

In children and in growing rats, NID produces intestinal malabsorption of carbohydrates by decreasing the activity of jejunal disaccharidases. Crypt cell surface membrane glycoproteins such as the secretory component (SC) of immunoglobulins are also depressed. To clarify, the mechanism(s) by which NID affects these intestinal functions, suckling rats made iron deficient in utero were studied at day 12 after birth. SDS-polyacrylamide gel electrophoresis of purified jejunal brush border showed that the protein band corresponding to lactase was virtually absent as was the incorporation of D-[$1C^{14}$]-glucosamine into lactase protein (3315 + 302 controls vs 140 + 10 cpm.mg prot.$^{-1}$, NID, $p < .001$). In a second experiment, rats with NID were studied after weaning (day 28). Measuring SC concentration into subcellular fractions of isolated jejunal cells, we found similar differences of SC content in the cytosol fraction (1.2 + 0.3 vs 5.65 + 1.2 µg.g.tissue^{-1}, $p < 0.05$) and in the brush border (0.7 + 0.1 vs 3.58 + 0.6 µg.g.tissue^{-1}, $p < 0.05$) between iron deficient rats and controls. Conclusion: These data indicate that NID affects the synthesis of intestinal brush border glycoproteins rather than the intracellular transport or the final membrane assembly of these proteins.

34 Rotavirus infection in hospitalized newborn infants. U.B. SCHAAD* and R. ZBINDEN*. Dept. of Pediatrics, Univ. of Berne, Switzerland.

Rotavirus (RV) gastroenteritis is found in 40% of our hospitalized pediatric pat. with acute diarrhea. This 3-year experience favorably compares to that of other centres in temperate climates. Nosocomial spread (fecal-oral, resp. droplets) of RV infection among pediatric pat. and medical personnel is well documented and is responsible for 7 % of our RV gastroenteritis cases. Recently, RV outbreaks in neonatal nurseries during RV infections in the community have been reported from Sidney, Melbourne, London and Washington: fecal RV excretion was found in 30-50% and remained asymptomatic in 70-92%. Since April 1983 we conduct a prospective surveillance study for RV infection in our referral 6-bed intensive and 8-bed special care nurseries. From each pat. fecal specimens on admission plus 3x weekly are examined for RV by ELISA technique (Rotazyme). From April to Sept. 1983 we observed RV in the stools of 31 (17.1%) of 181 neonates. In the majority of cases RV was detected between the 1st and 5th day of life and RV excretion lasted from 1 to 3 days only. Careful analysis of clinical and laboratory data revealed that all 31 neonatal RV infections were totally asymptomatic. During the first 6 study months recovery rates remained constant, but there was no community outbreak. Preliminary data during our RV season (winter months) indicate increased incidence also in neonates, but clearly RV related symptoms or signs were never detected. In our experience neonatal RV infection is rather common but of only short duration and extremely benign. It is suggested that both nosocomial spread and immunologic protection might explain these observations.