## 77 EARLY SCREENING OF INFANTS FOR HIGHER RISK OF THE SUDDEN INFANT DEATH SYNDROME.

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Infants at higher risk of the Sudden Infant Death Syndrome (SIDS) can be successfully detected and protected during the first year of life. From March 1977 to February 1988, 150 infants have been investigated for high risk of SIDS. There were 30 Near Miss infants,all successfully ressuscitated (Group I),35 siblings of SIDS victims (3 twins) (Group II),and 85 infants referred after an episode of pallor or cyanosis related to sleep (Group III).All were subjected to a mean of 2 night polygraphic recording of sleep stages,respiratory and heart rates as well as PtcO2.The infants from Groups I and III were also studied for a recognizable cause of the initial incident. 32 infants were considered at higher risk:every Near Miss (after exclusion of recognizable causes),one infant from Group II (abnormal polygraphic recording),and one from Group III(exclusion of recognizable causes and abnormal polygraphic recording). They were sent home with a cardio-respiratory monitoring.They all required stimulation by the parents for apnea or bradycardia,but there was no death.Among the 118 infants not considered at higher risk, none presented any incident.All 150 infants are well by now.

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Regulation of Breathing and Transcutaneous Oxygen in Infants during Sleep from birth to 6 months.

Polygraphic studies lasting 6 hours were made sequentially at 1 week,1,3 and 6 months in 14 infants (9 normal and 5 sibs of sudden infant death-SIDS). Heart and respiratory rate(HR,RR),transcutaneous oxygen($P_{tc}O_2$),and the incidence of apnoea,bradycardia and periodic breathing was compared at each age. HR and RR were higher in active sleep(AS)than in quiet sleep(QS)at all ages. In normal babies RR did not change with age but in the sibs of SIDS it was higher at 1 week and 1 month($P<0.05$). $P_{tc}O_2$ rose in the normal babies between 1 week and 1 month and in the sibs of SIDS between 1 week, and 3 months ($P<0.05$). There was no difference in mean $P_{tc}O_2$ between AS and QS at any age and the level was not different between groups at 1 month when the sibs of SIDS had a higherRR.The hypothesis that this group suffers chronic mild hypoxia is not supported(1). In normal babies between 1 week and 1 month the incidence of apnoea (3-5 secs) decreased in AS but increased in QS.Apnoea 6 secs was rare but most common at 1 week. No apnoea > 11 secs was recorded.Bradycardia (<100 bpm) accompanied 7% of apnoeic episodes at 1 week, this increased to 15% at 3 months but was absent at 6 months.Episodes of periodic breathing were most frequent at 1 month and then decreased progressively at 3 and 6 months. From birth to 6 months the mechanisms which control breathing develop with a separate time sequence for different sleep states.

1. Hoppenbrouwers T.T. et al Ped. Res. 10:425,1976.

## 79

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Circulatory adaptation to hypoxaemia during anaesthesia and surgery in newborn and two weeks old piglets

When piglets were subjected to anaesthesia and surgery (placing electromagnetic flow-probes around the ascending aorta) and, additionally in part, to hypoxia ($Pao_2$ lowered from 60-100 Torr [8-13.3 kPa] to 30-40 Torr[4-5.3 kPa] by changing $Fio_2$), a remarkably uniform response of the cardiovascular system could be observed: Soon after the beginning of the experiment cardiac performance increased by 12-35 percent ( $p < 0.01$) within 15-30 min. This was followed by a fatal decrease leading to cardiac failure accompanied by increasing metabolic acidosis.

In three groups mean survival time was approximately three hours: In the hypoxaemic newborns (n= 10), in both the normoxaemic (n = 8) and the hypoxaemic piglets two weeks of age (n = 8). The normoxaemic newborns (n = 8), however, survived significantly longer (416 ± 167 min; $p < 0.001$), and all the changes occured later. Survival time depended on pH decrease per hour in all groups and on the extent of the increase of heart rate in both hypoxaemic groups.

We conclude from our experiments that newborn and two weeks old piglets cannot sustain hypoxaemia of this degree longer than 30 min. Newborn piglets tolerate anaesthesia and surgery, not complicated by hypoxaemia, better than two weeks old animals.

## 80 SERUM FERRITIN(SF) MEASUREMENTS IN CHILDREN BY A NEW IMMUNORADIOMETRIC ASSAY

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The immunoradiometric assay(IRMA) for determination of SF can be ameliorated by use of a heterologous antibody(AB) system with a solid phase AB directed against liver and a $J^{125}$ labelled AB against placenta ferritin. We investigated 65 control subjects (age 1-21 yrs) without reduced Hb, MCH, MCV and transferrin saturation, 10 children with iron (Fe) deficiency anemia without increased sedimentation rate or serum transaminases (A),10 children with latent Fe deficiency(B), and 86 children with other disturbances of Fe metabolism: chronic renal failure under conservative (C) or dialysis (D) treatment and polytransfused non-renal anemia (E). Geometric mean(MG) and extreme limits of SF in controls increased with age: 23-102 µg/l at 1-5 yrs, 31-190 at 6-13 yrs, 39-292 at 14-20 yrs (MG of all: 66 µg/l). These values are higher than SF in normal children reported with former assays. SF in A was always lower than in controls (1-13 µg/l). Intermediate values were obtained in B(14-23 µg/l). SF showed a wide range in C (7-406 µg/l, MG 69) and was significantly higher in D (MG 553 µg/l) and E (MG 3509 µg/l). The highly sensivitive new IRMA enables a reliable differentiation between various states of Fe deficiency and overload.

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W. MÜLLER* and C.H.L. RIEGER* (intr. by: J. Brodehl). Effect of passive antibody on oral immunization with milk protein in human neonates. Kinderklinik der Medizinischen Hochschule Hannover, 3 Hannover 61, W.-Germany.

The effect of transplacental antibody on the immunresponse to Bovine Serum Albumin (BSA) normally present in infant formulas was assessed in 15 healthy neonates. Anti-BSA, determined by the Farr-assay using lng $^{*}$I-BSA-N/ml, was considered present if sera bound > 10% of $^{*}$I-BSA. If > 25% of $^{*}$I-BSA was bound, an antigen-binding-capacity (ABC) was calculated and expressed as ng BSA-N bound by 1 ml of serum. 7 infants (group I) with anti-BSA in their cordblood were compared to 8 infants without passive anti-BSA (gr. II). The development of anti-BSA (ABC) and IgG (mg/ml, gr. I and II combined) are shown in the table.

| | cord serum | | 4 weeks | | 8 weeks | |
|---|---|---|---|---|---|---|
| | ABC | IgG | ABC | IgG | ABC | IgG |
| gr. I | 33 ± 2 | 12.24 ± 3.77 | 17 ± 9 | 6.9 ±1.9 | 81 ±92 | 4.84 ±1.57 |
| gr. II | 0.0 | | 25 ±19 | | 211 ± 83 | |

The finding of a significantly higher ABC in gr. II (at 8 weeks $p=0.05$) suggests that passive antibody may modify the response to oral antigens although it does not prevent active antibody formation in early life.

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F. HANEFELD, TH. LENNERT, Dept. of Pediatrics, and G. STOLTENBURG, Dept. of Neuropath., Free University of Berlin, Berlin-West, F.R.G. CONGENITAL MYOPATHY IN LOWE' SYNDROME

The oculo-cerebro-renal syndrome described in 1952 by Lowe is an x-linked disorder charecterized by bilateral congenital cataracts, mental deficiency, growth retardation, muscular hypotonia, proteinuria, generalized aminoaciduria, metabolic acidosis and rickets. At present no basic biochemical lesion is known to explain this variety of abnormalities. We would like to report two cases in whom there is evidence of a muscular disorder as a possible cause of the hypotonia. Both cases showed the typical symptoms of Lowe syndrome, one was the second affected child. In addition to muscular hypotonia which improved over the years both patients show constantly elevated CPK levels up to 254 and 174 U/l respectively. Muscular biopsy taken at herniotomy revealed definite myopathy with small muscle fibers, variation in fibre size and single fiber necrosis. These findings explain the muscular hypotonia observed in our patients. Some investigators, however, were unable to demonstrate convincing myopathic changes in their cases. They considered the hypotonia as a neuropathic phenomenon probably resulting from an unknown metabolic derangement. Further neuromuscular studies of more cases are necessary to answer this question. It might, however, turn out, that Lowe syndrome is not an entity and that cases with myopathy form just a special group. If one considers this disease as a membrane or transport disorder the myopathy is of great interest.