

31 PANERO A., NODARI S., AGOSTINO R., MORETTI C., FERRO R., MENDICINI M., SAVIGNONI P.G. and BUC-CI G. Inst. of Paediatrics, State Univ. of Rome, Italy. Monitor-activated IPPV applied by nasal prongs for treatment of apnoea of prematurity.

An apparatus was devised allowing to shift from CPAP breathing by nasal prongs to nasal IPPV upon activation of the alarm of bradycardia (HR < 100/min) from the monitor attached to the patient. In 5 infants with apnoea of prematurity (bw 850-1200 g; g.a. 27-29 wks), HR, thoracic impedance and pressure in the respiratory circuit were continuously recorded, and consecutive periods with CPAP alone or with CPAP plus monitor-activated IPPV (MAIPPV) were compared. Termination of the spells was obtained by current procedures in the control periods, and following IPPV alone in most of the spells during MAIPPV periods. With MAIPPV, unquestionable immediate ventilation (according to thoracic impedance tracings) was obtained only in part of the spells. Nevertheless in 3 out of the 5 infants the following significant differences were observed with MAIPPV: shorter duration of bradycardia (\bar{m} 11-13 sec vs 19-23 sec); lower incidence of bradycardia > 20 sec (0-13% vs 43-62%); higher minimum HR (\bar{m} 68-75/min vs 53-58/min). In the remaining 2 infants no significant differences were observed.

32 A. Hrbek, P. Karlberg, I. Kjellmer, T. Olsson and M. Riha. Dept. of Pediatrics, University of Göteborg, Research Laboratory of Medical Electronics, Chalmers University of Technology, Göteborg, Sweden. EARLY DIAGNOSIS OF BRAIN DAMAGE AFTER PERINATAL ASPHYXIA BY MEANS OF EVOKED EEG RESPONSES.

The early diagnosis of brain damage is still an important problem and there is a lack of methods which can be used. Recordings of spontaneous electric activity provide no reliable information on the risk of damage after perinatal asphyxia. We try to use evoked EEG responses (ER) for these purposes. Preliminary results were already reported. **Material and Method:** This paper is based on 198 examinations in 90 neonates and infants with different degree of perinatal asphyxia. Visual evoked responses (VER) and photic driving (PD) were recorded in all of them, Somatosensory responses (SER) in 47 patients. More severe cases were examined several times. EEG was recorded bipolarly from corresponding projection areas and from vertex and averaged on a PDP 12 Digital computer. As stimuli light flashes and electrical pulses applied to n. medianus were used. **Results:** A number of alterations of ERS was observed. Most important were increased latencies and abnormal patterns of VERs, bad or absent PD and alterations of SERs. In order to express the findings in a quantitative manner a scoring system was devised. The total riskscore correlated well with the degree of asphyxia and with the prognosis. Almost all the infants with a permanent high riskscore developed symptoms of brain damage after a clinically silent period. **Conclusion:** Recording of ERS is a very useful objective method for early diagnosis of brain damage after perinatal asphyxia.

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Transitory thrombocytopenia in small-for-dates infants, and in newborn mice exposed to hypobaric hypoxia, cigarette smoke and CO gas inhalation during pregnancy.

In 31 small-for-dates infants platelet-counts were recorded during first 2 weeks after birth. In 23 (74%) platelet-counts fell below $100 \times 10^9/l$. Thrombocytopenia was significantly more frequent in small-for-dates infants of smoking mothers than in non-smokers. Mated female mice were exposed to hypobaric hypoxia (0.7 atm), cigarette smoke or CO gas during pregnancy. Smoke and CO caused HbCO levels of 4-10%. Intrauterine growth retardation took place in all three groups. Hypoxia caused increased hematocrits at birth, while decreased hematocrits occurred in all three groups day 11-15 after birth. Platelet-counts were decreased first 7-9 days after birth in all groups, followed by rebound thrombocytosis. Bone marrow megacaryocyte-counts increased more rapidly after birth (day 0-5) in the CO group than in controls, while hypoxia caused a later (day 11-15) appearing increase. Competition on common stem cells for erythropoiesis and thrombopoiesis may explain these findings. We suggest that CO toxicity impairs cell growth and proliferation during fetal life, and that deleterious effects of cigarette smoking on fetuses in part may be caused by CO toxicity.

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Usefulness of serum thyroid hormones determinations for the follow-up of treated congenital hypothyroidism.

Fourteen cases of primary congenital hypothyroidism aged 1 month to 9 years 6 months were followed with regular determinations of serum thyroid hormones. During 2 to 6 years follow-up, mean dose of desiccated thyroid gland powder was 63 ± 25 mg/m²/day (mean \pm S.D.). This dose corresponds to a mean serum thyroxine concentration of 82 ± 32 μ g/l. With a dose of 88 mg/m²/day (mean \pm 1 S.D.) all the thyroxine levels were above the lower limit of normals. With such a dose, a normal growth was assumed, as well as a catch-up growth in the patients who had a retarded growth. A good psychomotor development was observed in the early-treated patients. Serum l-tri-iodo-thyronine levels were not useful because of the wide fluctuations observed. Serum TSH levels were all under the lower limit of the normals with a dose of 88 mg/m²/day. In conclusion, regular measurements of serum thyroxine during therapy seem to be an adequate parameter for determining the dose of thyroid gland extracts adequate for substitution.

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Disordered Intestinal Function in Glycogen Storage Disease. Chronic diarrhoea is a recognised feature of glycogen storage disease (GSD), but the mechanism is not known. The in vivo absorption of glucose and 3-O methyl glucose (3MG) has been studied using a steady state perfusion technique of the jejunum with simultaneous recording of the transmural potential difference (PD). Net absorption of 2mM and 56mM glucose was reduced in a patient with Type 1 GSD (0.43 ± 4.94 μ moles/min/cm jejunum) compared to a control group (0.55 ± 0.02 & 8.85 ± 1.07) but absorption of 56mM 3MG did not differ from that of the control group. Water was secreted during perfusion of 2mM glucose but with 56mM glucose and 3MG water absorption occurred. PD during glucose perfusion (2mM & 56mM) was less (-4.2 & -7.8 mV) than in the control group (-5.1 ± 0.1 & -8.9 ± 0.4). During 3MG perfusion PD was similar to controls. Glycogen metabolism in jejunal mucosa was studied histochemically and the in vitro uptake of glucose was measured. Histochemical studies showed the presence of glycogen and lack of glucose 6 phosphatase activity in the jejunal mucosa of affected patients whereas in the control group glucose 6 phosphatase activity was present but no glycogen could be detected. In vitro uptake of glucose (2.5 mM) was also reduced, the tissue/medium ratio was 3.98 ± 15.88 (controls 36.83 ± 4.8). These studies show that in type 1 GSD there may be malabsorption of glucose, and at low luminal concentration of glucose a secretory state may exist.

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BILE SALT SECRETION IN CHILDREN WITH FLAT JEJUNAL MUCOSA.

The mechanism by which intestinal hormones are secreted and active during meals is poorly understood. This study deals with the role of intestinal absorption on hormone release. The bile acid secretion (J_{BS}) and bile acid concentration [BS] has been measured under intraduodenal milk infusion (4ml/min m²) and intravenous CCK (2 μ /kg) injection in children with normal jejunal absorption and with jejunal malabsorption; i.e. 10 "control" children (group I), 8 children with flat jejunal mucosa (group II), 3 children previously in group II, with normal jejunal histology under gluten free diet (group III). Results: with milk infusion, J_{BS} (in μ mole/min m²) is significantly lower (P < 0.01) in group II (25 ± 3) and in group III (19.5 ± 1.4) than in control group (46 ± 6). Similar differences are observed with [BS] (group I: 6.5 ± 0.9 μ mole / ml; group II: 3 ± 0.4 μ mole / ml; group III: 2.7 ± 0.3). After IV CCK the response in group II is heterogeneous; 6 children have a low response (from 2.04 to 41.4) while 3 children have a response within the control range (group II: from 59.9 to 72.37, "control": 52 ± 2). These results suggest that bile salt secretion is impaired in children with flat jejunal mucosa and also in children with coeliac disease and normal jejunal histology under gluten free diet. In conclusion, the CCK intestinal release and efficacy may not be exclusively under jejunal absorption control.