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A SIX-YEAR FOLLOW-UP OF <1000 g BIRTH-WEIGHT PREMATURES
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Between January 1980 and December 1985 a number of 28,975 live-born were delivered at Kaplan Hospital. The group of the very very low birth-weight infants (VVLBW) 501 to 1000g included 157. Fifty-four of them were discharged home. A survival of 10.4% (7/67) between 501-750g and 52.2% (47/90) between 751 and 1000g. During the first year after discharge 3 infants died. The mean weight of the consecutive 51 VVLBW infants enrolled in this study was 883g with a mean gestational age of 26.7 weeks. The developmental quotient (DQ) was determined according to Gesell's charts between 24 to 72 months of age. The outcome of the population was evaluated according to a "functional handicap" score, based on combined DQ, degree of cerebral palsy and sensorineural impairment. Functional handicaps were found in 21% of the survivors (11% moderate to severe, 10% mild). Our results correlated with the better survival prognosis for the 751 to 1000g group without significant rate of morbidity, among the survivors. The mean DQ of 90 and low rate of functional handicap among the survivors seems to justify the intensive care approach for this very high-risk population.

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COMPARISON OF OUTCOME IN VERY PRETERM AND VERY LOW BIRTHWEIGHT INFANTS AT THE AGE OF TWO AND FIVE YEARS.

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In the nationwide cohort (1338 liveborn infants <32 weeks and/or <1500 g), infant mortality was 27.2 % (n=364). At the age of 5 years 944 children could be traced; of these, 911 were examined during a home visit. Preliminary results of 542 children show a major handicap in 24 children (4.4%); minor handicap was present in 25 children (4.6%). Comparison to the results of the follow-up study at 2 years in the same children (4.2% and 10.0% respectively) showed a similar major handicap rate and a lower minor handicap rate. However, shifting between the various subgroups (normal, minor and major) had occurred, a more favourable outcome was seen in 52 children and a less favourable in 23 children. Possible explanations of such shifts will be discussed.

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LONG-TERM EPIDEMIOLOGICAL AND CLINICAL STUDIES OF INSULIN-DEPENDENT DIABETES MELLITUS
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This study presents the clinical and epidemiological analysis of data from 373 children representing all cases of insulin-dependent diabetes mellitus (IDDM) diagnosed during the first 16-yrs of life in the Gdańsk and Elbląg regions. Investigations were performed on the basis of a registry of new cases conducted from 1970 up to date. An increase in the incidence of IDDM was observed during these years. At the beginning of the 1970's 2.6 new cases were noted per 100000 population per year in comparison to 6.7 per 100000 population in 1988. An analysis of the incidence of IDDM in respect to sex, age, residence, season, family history of diabetes mellitus and infection prior to onset was performed. The increase in incidence was especially noted in the youngest age group (<3 yrs). Our observations suggest that the clinical course of IDDM in this group is different. The prevalence of IDDM among children from Gdańsk region was much higher than in the Elbląg region.

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DIETARY TRIGGERS OF IDDM. - Julio M. Martin, Moira Glerum, Brian H. Robinson. - Research Inst. The Hospital for Sick Children, Toronto, Canada.

Higher incidence of IDDM was reported in children exposed to cow milk early in life vs. those breast-fed for longer periods. Spontaneously IDDM rodents are also protected by elimination of cow milk proteins (Prot) in the diet, mainly β -lactoglobulin (LG) and bovine serum albumin (BSA), when such restriction occurs at weaning. We found that antibodies (Ab) to BSA (but not to LG) cross react with a BB-rat islet cell membrane prot. (Mr=69Kd) as demonstrated by Western blotting and by Fluorography when extracted radioactive labelled islet-cell prot. were immunoprecipitated and separated by gel-electrophoresis. The precipitate increased in cells pre-exposed to interferon- γ . Comparative aa. sequences of human, rat and bovine SA. showed clear differences in the latter vs. the others between residues 138-166, which could serve as epitopes. Overlapping this region (aa.157-175) is a homologous area between BSA and the primary structure of β -subunits of prot. Ia, and DQ and DR (aa.50-79). Corresponding to aa-57 of DQ antigens, human albumin has an Asp. residue while BSA has an Ala. Similar substitutions occur in the Ia- β prot. of IDDM mice and rats vs. normal controls. Hypothesis: Absorption of milk BSA early in life triggers an immune response by Ab. that cross-react with MHC Class II antigens as a typical "mimicry" event.

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URINARY ALBUMIN EXCRETION IN DIABETIC CHILDREN IN RELATION TO AGE, SEX AND THE DURATION OF DISEASE.

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Urinary albumin excretion was determined by the RIA method in 183 diabetic children of age between 2 and 18 years (mean age 12.63 $\pm$ 3.99 years). Daytime (6⁰⁰-18⁰⁰) and night (18⁰⁰-6⁰⁰) fractions of urine were collected separately. The children were divided according to the time of duration of diabetes into five groups: I: below 1 year, II: 1-5 years, III: 5-9 years, IV: 9-12 years, and V: above 12 years. Microalbuminuria (albumin excretion higher than 20 ug/min) was observed in group IV (34.8 $\pm$ 10.1) and in group V (125.8 $\pm$ 39.8). No relation was found between urinary albumin excretion and age or sex of children. Also there was no difference related to the time of the onset of diabetes (before or after age of 11 years). There was a strong correlation (r=0.534, p 0.001) between urinary albumin excretion and the duration of the disease. During the daytime the urinary albumin excretion was significantly higher (p 0.02) than that during the night, irrespectively to the duration of diabetes.

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CORTICOSTEROID TREATMENT AFFECTS LH BIOACTIVITY IN PUBERTAL RENAL TRANSPLANT PATIENTS

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Delayed onset of pubertal development is common in chronic renal failure (CRF). We investigated nocturnal pulsatile immunoreactive (iLH) and bioactive (bLH) LH secretion between 8 pm and 7 am in 19 pts who had received a renal transplant. Age ranged from 11.7 to 23 yrs; all stages of puberty were represented. Immunosuppression was maintained by corticosteroids and cyclosporin A and/or azathioprin. The methyl-prednisolone equivalent dosage ranged between 1.2 and 14.7 mg/m²day. iLH and bLH were determined by RIA and the mouse Leydig cell assay respectively. Data were analysed by PULSAR and Cluster analysis programmes. LH peak frequency increased with puberty stages to a maximum at PH4. Peak areas were correlated exponentially with age and TW2 bone age (p=0.02). Peak amplitudes did not change significantly. The mean bLH/iLH ratio was 1.5 $\pm$ 0.4. It did not change with age, stage of puberty or serum creatinine. However, we found a strong inverse relationship with prednisone dosage (r= -0.72, p < 0.002). As LH bioactivity is modulated by enzymatic glycosylation and sialisation, we suggest that corticosteroid treatment may interfere with these processes.

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