61

COST-BENEFIT ANALYSIS OF NEONATAL CARE

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Costs of neonatal care in the County of Vestfold 1980-84 (level II neonatal unit, 15% admitted from an unselected population averaging 2087 deliveries a year) were US$ 0.8 million a year (1984 exchange) (including costs of level III intensive care and transportation), 1.6% of the county's total costs for hospital services. Costs per treated patient were on average US$ 2443. Salaries accounted for 82.2%, running expences 13.5%, and equipment 2%. Epidemiological data on neonatal mortality and handicaps showed a net gain of 25 infants with intact survival 1980-84 compared to 1970-79. Costs of treatment for these 25 patients (calculated as the 5 most expensive patients each year 1980-84 with intact survival) were on average US$ 28409, rehospitalization costs during the year after birth included (6.7% of the expenditures). Total lifetime income and taxes were calculated to 21.2 and 3.1 times treatment costs. Progress in neonatal care 1970-84 in our county has caused considerable medical gains, with a strongly positive economic benefit.

62

INCIDENCE OF AUTOANTIBODIES IN NORMAL CHILDREN

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Very few data are reported on the incidence of autoantibodies (AA) in normal children. We have studied the incidence of 14 AA in a total of 268 apparently normal children (151 males and 117 females; age range: 2 months-14 years with a homogeneous distribution for each year of age). Antinuclear (ANA), anti-mithocondrial (MA), anti-ribosomal (ARA), anti-smooth muscle (SMA), anti-reticulin (RA), anti-double stranded DNA (dsDNA), anti-gastric parietal cell (PCA), anti-intestinal epithelial cell (IECA), anti-liver/kidney microsomal (LKM), islet cell (ICA-IgG) and complement-fixing islet cell (CF-ICA) antibodies were determined by indirect immunofluorescence; rheumatoid factor (RF) was detected by latex agglutination; anti-thyroglobulin (TgA) and anti-thyroid microsomal antigen (MsA) antibodies were detected by passive hemagglutination. 41 children (22 males and 19 females) were positive for at least one AA, usually in low titer; two were positive for 2 AA. None of these children had a personal or family history of autoimmune diseases. The percentage of children positive for each AA was as follows: ANA 3%, SMA 2.6%, RA 2.6%, MA 1.1%, RF 0.6%, ARA 0.4%, PCA 5.2%, MsA 1.3%. Anti-dsDNA, IECA, LKM, ICA, CF-ICA and TgA were not detected in any sera. Fifteen of the 41 positive children were checked again for the presence of AA two years later; 6 (40%) were still positive, always for the same AA, without major differences in titer. Our results show that the incidence of serum AA in normal children is similar to that reported in young adults. In more than half this positivity appears to be a transient phenomenon; the possible significance of the persistence of AA in some apparently normal children remains to be defined.

63

CROSS REACTIVITY WITH HUMAN BASOPHILS OF MONOCLONAL ANTIBODIES RAISED AGAINST MEMBRANE COMPONENTS OF RAT MAST CELLS OF THE RBL-2H3 LINE. C. Geller-Bernstein*, A. Berebi*, E. Ortega**, A. Licht** and I. Pecht**. Kaplan Hospital*, Weizmann Institute**, Rehovot, Israel.

Leukocyte samples of 16 atopic and 14 non-atopic children were enriched in their basophils by single step Ficoll-Pague centrifugation to a relative concentration of 10-15% (Miroli, et al. 1986). Aliquots from each of these basophil preparations were incubated with the following monoclonal antibodies (mAbs): F4 and H10 shown previously to be specific for the high-affinity Fcε receptor present on the RBL-2H3 cells; G63 which recognizes a membrane protein different from the Fcε receptor and shown to cause inhibition of IgE mediated degranulation of RBL cells; B17 specific for a glycolipid present in the plasma membrane of RBL-2H3 cells and modulates their degranulation. In addition a monoclonal murine IgE was used to probe the occupancy of Fcε receptors. Following incubation, the binding of the mAbs was monitored by fluorescently labeled rabbit anti-mouse antibodies and the samples were analysed by the Fluorescence Activated Cell Sorter (FACS 440). The results show that all four mAbs also bind, though to different extents, to partially purified human basophils of both atopic and non-atopic children.

64

Hyper-peptiduria in Williams syndrome

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Six patients with classic Williams syndrome and the behavioural "cocktail party" manner were investigated for glycoprotein bound peptides in 24 h. urines (1). Pathological chromatographic patterns were obtained in all 6. 4 had peptide increases above the normal level. Average for Williams syndrome patients was 20.9 ± 10.5 (n=6). The normal is 7.9 ± 2.4 μmoles (n=7.5) hydrolysis released amino acids. The peptide increase may entail a dietary etiology and partly explain behavioural changes (2).

1. Reichelt, K.L. et al (1986) Biol Psychiat 21, 1279-1290
2. Reiss, A. et al (1985) J Pediatrics 106, 247-249

65

PNEUMOCYSTIS CARINII PNEUMONIA IN NEONATES

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Pneumocystis carinii may be one of the etiologic factors of pneumonia in neonates. Of 258 neonates enrolled in a prospective study of neonate pneumonia 57 (22%) had evidence of Pn.c. infection. Diagnosis was based on patients history, clinical signs, chest roentgenogram, blood gas examination, serologic tests detecting the patients specific fluorescent IgM and IgG antibodies and cytologic examination of tracheal lavage performed in 14 cases showing 100% correlation with the serologic tests. Patients were hospitalised at mean age of 5 days (range 1-20) and their illness was characterised by its afebrile course, presentation in crisis with severe respiratory distress, bilateral pulmonary infiltrates with hyperaeration. Treatment with Lomidine or Trimethoprim-sulfamethoxasole, in most severe cases with both drugs, was associated with rapid improvement. None of the patients died. These results indicate that Pn.c. may be an important cause of pneumonitis in neonates. An early diagnosis results in full therapeutical success. Considering the age of the patients congenital Pn.c. pneumonia cannot be excluded.

66

PURINE NUCLEOTIDE SUPPLY OF THE HUMAN TROPHOBLAST

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Feto-placental unit requires large amounts of nucleotides and nucleic acids due to its rapid growth and metabolic rate. We studied purine nucleotide synthesis in a highly enriched population of trophoblastic cells from normal first and third trimester placentae, obtained with collagenase digestion and density gradient. De novo synthesis was measured as incorporation of ^{14}C-formate and reutilization as incorporation of ^{14}C-adenine (Ade), -hypoxanthine (Hx), and -adenosine (Ado). Incorporation of formate was significantly ($p<0.01$) less in third trimester cells. Ade incorporation was an order of magnitude higher than that of formate, and significantly ($p<0.001$) higher in first than third trimester cells. Hx incorporation did not change as a function of gestational age. High (10 mM) extracellular inorganic phosphate did not enhance Hx phosphoribosylation. Both Ado phosphorylation and deamination increased with concentration. High Ado (60 uM) was more efficiently utilized in first trimester cells. Concl.: 1) major pathways of purine nucleotide synthesis are functional in human trophoblast throughout gestation, 2) contribution of reutilization to the synthesis appears larger than that of de novo pathway, 3) rate of nucleotide and nucleic acid synthesis decreases with gestational age, 4) hypoxanthine may be the major precursor utilized in trophoblastic purine nucleotide synthesis.