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URINARY PROSTAGLANDIN EXCRETION IN SEVERE MALNUTRITION.

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With the aim to study the relationship between malnutrition, essential fatty acid (EFA) intake, renal production of prostaglandin (PG's) and renal function, we have measured urinary PGE2 and PGF2α by RIA, urine and serum electrolytes, creatinine clearance, plasma renin activity (PRA) and EFA in serum phospholipids by gas chromatography in 15 children aged 1 to 3 years and severely malnourished (marasmic kwashiorkor, wasting index $<80\%$).

Results: ($\bar{X} \pm$ SEM):

	n	E2 ng/m2/d	F2α	UV ml/d	UNa mEq/d	Ccreat ml/'/1.73	PRA ng/ml/h	C18:2 %	C20:4 %
A)	13	155.0	701.2	303	3.8	47.8	54.3	21.66	5.12
		78.0	233.6	75	1.3	7.1	12.7	2.34	0.75
B)	9	99.8	456.9	498	24.0	66.1	20.9	21.25	4.84
		27.7	124.8	51	4.8	11.2	7.0	2.21	0.53

A = at admission; low EFA intake according to dietary history; B = after 10 days of equilibration diet containing 15% of calorie intake as linoleic acid. Urinary PG's were low in 5 patients (E2) and elevated in 2 (E2 and F2α). No correlation was found between PG's and UV, UNa or PRA. A small but significant correlation was found between log PG and Ccreat. In severe malnutrition EFA intake seems not to affect PG synthesis at the renal level.

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STOPPING MEDICATION IN EPILEPTIC CHILDREN. A STUDY OF RISK FACTORS RELATED TO RECURRENCE.

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The series included 70 children who had experienced at least 2 seizures before 12 years old, excluding febrile seizures, neonatal seizures or seizures ocurring during a metabolic or infectious insult to the SNC. The children were at least 2 years seizure free and the drug (s) was discontinued over a 3 month period (for each drug). Each child had a complete neurological and psycological assessment, and an EEG was performed every 6 months. Focal neurological signs, an I.Q. less than 70 and epileptic abnormalities on the EEG were considered as an abnormal parameter for statistical analysis. The seizures were classified as grand-mal (GM), absences (PM) and partial on a clinical basis. 20 children (28,5%)experienced a recurrence. 75% of them had the seizure during or less than 6 months after withdrawal of the anti-epileptic drug. The factors evaluated for their relation with recurrence were: Age at onset of seizure (before and after 3 y), types of seizure, neurological and/or psycological abnormalities, number of seizures before control and EEG abnormalities. Those which reached statistical significance were: a) more than 10 seizures before control ($0{,}04 > p > 0{,}025$); b) at least 1 abnormal EEG before drug withdrawal ($0{,}02 > p > 0{,}01$); c) neurological and/or psycological abnormalities ($0{,}01 > p > 0{,}002$); d)association of GM seizures with other types of seizure ($0{,}002 > p > 0{,}001$). 14 children of the recurrence group (70%) had 2 or more of the above risk-factors while 36 (72%).of the non-recurrence group had none or only one.

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COMPARATIVE STUDY BETWEEN THE METHODS OF DOUBLE DIFFUSION IN GEL AND RADIAL IMMUNODIFFUSION OF IgA AND IgM IN CORD BLOOD.

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In order to find a simple and low cost method that could be used for routine determination of IgA and IgM in cord blood for screening of congenital infections, we studied comparatively the method of Double Diffusion in Gel Ouchterlony (DD) with the method of Radial Immunodiffusion-Mancini (RID), commonly utilized. Cord blood samples were obtained from 85 newborns and IgA and IgM were determined concomitantly by the two methods.The preliminary results showed 11 cases where the determinations of IgM of IgA were negative by RID but positive by DD with high titles. A repeated dosage by RID with serum diluted by half or using immunodiffusion plates for higher concentrations, showed positiveness for these immunoglobulins, proving therefore, the importance of double diffusion in detecting falsely negative results obtained by the radial immunodiffusion method. In 6% of the cases, IgM values obtained by RID were higher than 20 mg/dl were detected from title 1/2 up, corresponding to 31.76% of the studied population. For screening of congenital infections, IgM titles equal or superior to 1/2 by DD should be complemented quantitatively by RID. Statistic analyses showed a parallelism between these two methods.Taking into account its low cost and simplicity, the method of double of diffusion in gel is recomended for the routine determination og IgA and IgM in cord blood.

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CHANGES IN INTESTINAL PERMEABILITY DEMONSTRATED BY LOW MOLECULAR WEIGHT POLYETHYLENE GLYCOL (PEG) POLYMERS.

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The purpose of this study was to compare the changes in intestinal permeability produced by villous atrophy with those produced by the administration of a cytotoxic drug. PEG (mol wt 200-600,ethylene units 5-13), a marker for intestinal permeability, was administered orally as an isotonic solution at a dose of 172 mg/kg to 26 children with celiac disease (CD) (ages 1-18 yr) and to 6 children (8-12 yr) treated for leukemia with 20 mg/m^2 of methotrexate (MTX). Urine was collected for 6 h. Children with CD were studied at the time of presentation (P), while on a gluten (G)-free diet (GFD), and/or when challenged with a G-containing diet (GCD). Children with leukemia were tested before and after MTX. PEG polymers were isolated, derivatized, and analysed by gas chromatography (J Lab Clin Med 107: 290,1986). The 6-h urine % recovery of each polymer and the length of the polymer whose recovery was maximal (PCDO) were determined. The theoretical ethylene unit length of the polymer whose recovery was 50% of the polymer which was recovered maximally ($N_{1/2}$) was calculated by a curve-fitting program. Normal $N_{1/2}$) $\geq$ 12. Mean $N_{1/2}$ for children with CD at P was 7.3 ± 3.7,on GFD: 12.8$\pm$0.6,and on GCD:11.4 $\pm$ 2 ($p < 0.05$) indicating significantly less recovery of the longer polymers at P compared to GFD and GCD. No relationship was found between G intake and PCDO. In children treated with MTX, a mean increase in PCDO of 127% (range 49-195, $p = 0.05$) was observed following the ingestion of the drug compared to pre-treatment levels. Conclusion: This study shows that in cases of intestinal villous atrophy permeability to the longer chain PEG polymers decreases while treatment with MTX produces an enhanced permeation to all of the PEG polymers.

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HEMATURIA IN CHILDREN: Metabolic Evaluation.

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After excluding nephrological and urologic disorders, recurrent hematuria in children may be related to metabolic disorders. We postulate that the mechanism for hematuria caused by metabolic disorders is increased calcium oxalate crystallization causing tubular epithelial injury. We studid 17 children aged 4 to 14 years (10 M, 7 F) with recurrent hematuria and normal uroradiological studies at the Children's Hospital Ricardo Gutierrez and the Metabolism Section of the Institute of Medical Investigation in Buenos Aires, Argentina. Each child was studied in the Hospital on a stable and neutral diet containing 100 mEq of sodium, 800 mg of phosphorus, and 1200 mg of calcium daily. On the third and fourth day, 24-hour urine samples were collected. On the fifth day fasting venous blood and a 2-hour urine samples were studied for calcium, magnesium, uric acid, phophorus, creatinine, sodium, potassium, oxalic acid, and alkaline phosphatase. The results of the 24-hrs. urine collection (expressed as X + S.D) in mg/kg/24-hrs. were: Ca 4.57 + 0.33, uric acid 13.4 + 2.5, Mg 1.2 + 0.1, and oxalic acid. 68 + 0.5. The UCa/UCr ratio was 0.24 + 0.13 for the 24-hrs. urine samples and 0.06 + 0.03 for the 2-hrs. fasting samples. A metabolic abnormality was detected in 82% (14/17) of the subjects. Hypercalciuria was detected in 47% (8/17) with 35% (6/17) of the renal type and 12% (2/17) of the tubular type. Hypomagnesiuria was detected in 29% (5/17) and both hypercalciuria and hyperuricosuria were seen in one subject (6%). No metabolic abnormality was found in the remaining 18% (3/17). We conclude that children with recurrent hematuria after the exclusion of diseases should be evaluated for metabolic abnormalities as a cause of recurrent hematuria.

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IMMUNOLOGICAL EVALUATION "IN VITRO" OF CHILDREN WITH SEVERE BRONCHIAL ASTHMA.

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Several studies relate the hyper IgE observed in atopic patients to a decrease in the T suppressor cell population. We studied 27 atopic children, aged 2y7m to 14 years, with severe perennial bronchial asthma Peripheral blood was obtained after 1 day without any bronchodilator medication and after 7 days without corticosteroids. IgG, IgM and IgA were determined by radial immunodiffusion and IgE by enzyme immunoassay Total T lymphocytes, T suppressor cells and T helper cells were determined using monoclonal antibodies (ORTHO, OKT_3, OKT_4, and OKT_8). Lymphocyte cultures were stimulated with phytohemaglutinin (PHA) and the results expressed as s stimulation index. Our data, compared to our normal values showed: Normal IgG, low IgA, hyper IgM and hyper IgE (this is the usual pattern observed in our asthmatic population). The percentages of OKT_3, OKT_4 and OKT_8, as well as the ratio $OKT_{4/8}$ were within normal limits. The response of the peripheral lymphocytes to PHA was normal in the presence of autologous and homologous plasma. Based on these results, we concluded that there are no important immunoligical changes in atopic asthmatic children and that there is no need for the use of immunodulating agents in the treatment of bronchial asthma in children.