

**11** BONE MINERAL DENSITY (BMD) EVALUATION IN PATIENTS WITH LONG-TERM GLUCOCORTICOID THERAPY: EFFECT OF ORAL CALCIUM AND VITAMIN-D. H.R.Cassinelli, C.A.Mautalen, C.Bergadá. CEDIE, Hosp.de Niños "R.Gutiérrez"; Lab.de Osteopatías, Hosp.de Clínicas; Centro de Osteopatías, Bs.As., Argentina.

Excess glucocorticoids induce osteoporosis. The effect of calcium and vitamin D on its prevention is uncertain. To clarify this issue we studied 2 groups of patients: group 1 (G1), n=8, who received methylprednisone (MP), vitamin D (ergocalciferol) 2900 + 1700 U/day\* and calcium 1.0±0.5 gr/day\* for 3.65±3.74 years\* (r: 0.4-9 years); group 2 (G2) n=15, patients received MP alone for 3.78±2.46 years\* (r:0.5 to + 10 years). BMD of radius shaft (RS) and lumbar spine (LS) (L2-L4) was determined by single and dual photon absorptiometry. Compared with normal controls, a significant reduction in BMD was observed in the RS of G2, 2 score was -0.8±1.02\*, p<0.01 and in LS of both groups, G1:-1.86±2.56\*, p<0.05; G2:-2.0±1.67\*, p<0.0005. A negative correlation was found both in G1 and G2 between BMD (Z score) of RS and LS, and the duration of treatment. Furthermore, during the first 3 years of treatment, only G2 showed a significant decrease of LS BMD; when treatment lasted more than 4 years, G1 also showed a reduction of RS and LS BMD. Long-term glucocorticoid therapy induces bone demineralization, principally affecting trabecular bone. This could be prevented by adding calcium and vitamin D but benefits occur only during the first 3 years of treatment.

\*= $\bar{X}$ ±SD

**12** HISTOLOGICAL MATURATION OF THE BRAINSTEM AND CEREBRUM IN THE SUDDEN INFANT DEATH SYNDROME (SIDS). M.E.Cordero, S.Benveniste, J.A.Nuñez, M.E.Trejo, M.V.Vásquez, R.Prado. Depto.de Morfología Experimental, Fac.de Medicina, Universidad de Chile, Santiago de Chile.

Sudden infant death syndrome (SIDS) has been defined as the sudden unexpected death of an infant during sleep and in which a thorough postmortem examination fails to demonstrate a cause of death. However, abnormal patterns of respiration, sleep and heart rate activity have been reported. Besides higher values of dendritic spine density have also been observed. We studied the histological maturation of neurons from the nucleus Principal Olivary, Hipoglossus and pontine and the pyramidal cells of the motor cortex of 3 infants diagnosed as SIDS and 2 non-SIDS (pneumonia) of 1 to 2 months of postnatal age. A quantitative Golgi Cox analysis of the total mean dendritic arborization was performed. Lower values of dendritic density in SIDS brainstem compared to controls were observed while no differences in basilar dendritic arborization of pyramidal cells of SIDS and non SIDS was detected. Our findings may indicate an immature developmental pattern in SIDS brainstem, suggesting that a noxious influence alters the ontogeny of brainstem development but does not alter ontogenesis of the cerebral cortex. The quantitative differences in dendritic density could be considered as an anatomical substrate of brainstem dysfunction in the multifactorial pathogenesis of SIDS.

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**13** HEART RATE AND ITS VARIABILITY IN SUDDEN INFANT DEATH SYNDROME (SIDS) VICTIMS. P.Peirano (1,3), B.Cauchemez (2), N.Monod (1). (1) INSERM. (2) Hospital Lariboisier, Paris, Francia. (3) INTA, U. de Chile, Santiago.

To evaluate whether abnormalities of heart rate (HR) control may be detected in SIDS victims prior to their death. We retrospectively analysed Holter and cardiopneumographic recordings of 19 infants (11 m, 8 f) aged 2.3 +/- 1.5 months who subsequently died of SIDS at the age of 4.2 +/- 2 months. Each SIDS victim was matched for gestational and postnatal ages and reference diagnosis with 3 control infants without SIDS after at least one year follow up. Nine hours of recording (9 pm to 6 am) were processed in terms of mean HR and HR oscillations (osc). Mean HR was higher in the SIDS group (141 +/- 14 vs 135 +/- 15 bpm, p<0.05, covariance analysis with age). HR short oscillation respiratory sinus arrhythmia: RSA) were lower in the SIDS group (3.35 +/- 0.59 vs. 3.65 +/- 0.61, p<0.05, covariance analysis with age). HR long oscillation (20 to 32 respiratory rate (RR) were similar in both infant groups (3.20 +/- 0.66 vs. 3.21 +/- 0.53) and were not correlated with age. The mean HR was modulated in the control group during the night, being lower at midnight and higher at 9 pm and 5 am. The mean HR in the SIDS group remained constant throughout the night. In an infant population at higher than normal epidemiological risk for SIDS, infants who subsequently died differed from those who did not die because the mean HR was higher in SIDS victims, and the short oscillation were absent. These results reflect a decrease in vagal tone in SIDS victims which can be absolute or relative to an increase in sympathetic tone. The fact that these differences between SIDS victims and control infants were especially observed in the middle of the night may express an absent or disturbed circadian organization of the waking-sleep rhythm and/or of the circadian modulation of the autonomous nervous system tone in SIDS victims.

**14** MACRONUTRIENT BALANCE IN ACUTE DIARRHEA OF INFANTS. G.V.Venegas, C.Castillo-Durán, M.Henríquez, J.C.Villalobos, L.E.Gatica. Pediatric Dept., Fac.of Medicine, Univ.de Concepción and INTA, University of Chile.

OBJECTIVE: To study fecal and urinary losses of nitrogen and fat, in milk fed infants admitted for acute diarrhea. MATERIALS AND METHODS: metabolic balances were performed in 31 male infants (2 to 11 mo), on days 1-2 and 6-7 after admission in the hospital. All had history of less than 3 days of diarrhea and dehydration. They were fed increasing volumes of diluted powdered cow's milk (8% w/v) plus 5% sucrose and 3% maltodextrine and rehydrated only with an oral solution (60mEqNa/1); no antibiotics were used.

	BALANCE I		BALANCE II	
	Rotavirus (N=16)	E.Coli (N=15)	Rotavirus (N=16)	E.Coli (N=15)
Fecal Exc.	131±61.2	141±82.7	53±24.3	* 70±16.4
Nitrogen (mg/kg/day)				
Urinary Exc.	189±72.8	175±66.1	176±49.8	165±48.7
Fat Fecal Exc.	1.7±1.4	1.7±0.4	0.3±0.4	0.7± 0.7

In Balance I 13 out of 16 with rotavirus and 8/15 with E.Coli had negative N Balance (p<0.05). In Balance II infants with diarrhea due to E.Coli presented higher N fecal excretion (\*p<0.025). CONCLUSIONS: Fecal losses of nitrogen and fat are similar in rotavirus or E.Coli diarrhea during the first stage. Fecal N losses are higher with E.Coli during the recovery of the illness. In the first stages of acute diarrhea nitrogen balance is more frequently negative with rotavirus than with E.Coli, with increase of both fecal and urinary N losses.

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**15** FRAGILE X (X FRA) SYNDROME: THE USEFULNESS OF A CLINICAL SCORE IN PATIENT SELECTION. M.Torrado, S.Tenembaum, C.Barreiro, N.Fejerman, Lic.J.Herrera, L.Bin, SAMIC. Garrahan Hospital-Buenos Aires, Argentina.

X Fra is the second cause of mental retardation (M.R.) after Down's Syndrome, representing 50% of the X-linked cases. The X-Fra phenotype has been described and common features are being defined in multicentric programs. We studied 46 children presenting 4 or more of the signs in a score: 1)Family history suggestive of X-linked pathology; 2) Height and cephalic circumference on P50 or more; 3)Large ears; 4)Prominent ears; 5)Long face; 6)High palate; 7)Calluses on the dorsal aspect of hands; 8)Joint hiperlaxity; 9) Depressed sternum; 10)Mitral prolapse; 11)Macroorchidism; 12)Poor visual contact; 13)Hiperactivity; 14)Stereotypes behavior; 15)Self-agression; 16)Disorders language development; 17)Variable degrees of M.R. We assign one point for each item. Twelve children, aged 3 to 14 years resulted X-Fra+ in lymphocyte cultures with added Fudr (50 cells analyzed). Negative X-patients showed less than seven signs of the score, whereas twelve or more signs were found in X-Fra+ children. Each patient was evaluated by geneticist, a neurologist and a neuropsychologist. The future aims of this program are: A) To identify X-Fra+ cases in a population of M.R. children without known etiology. B) To select through the score those patients to be evaluated cytogenetically. C) To provide genetic counselling to mothers and female relatives at risk.

**16** NUCLEOTIDE-ENRICHED MILK AND DIARRHEAL DISEASE IN INFANTS. J.Espinoza, M.Araya, S.Cruchet, I.Pacheco, O.Brunser. INTA, Universidad de Chile, Santiago, Chile.

Maternal milk contains factors that protect against diarrhea, among than nucleotides. The effect of a nucleotide-enriched formula on the incidence, duration and etiology of acute diarrhea was evaluated in 139 infants (G1). Children were admitted throughout the year and each child was surveyed for three months. The control group (G2, N=145) received the same formula without the added nucleotides. G1 and G2 children suffered from diarrhea 2.7% and 4.2% of the follow up period, respectively. G1 had significantly less episodes of diarrhea and total number of days with the disease (p<0.05 and p<0.0000, respectively). However, the duration of episode was comparable. Cases of persistent diarrhea were similar in both groups (N=8). Search of etiologic agents (bacteria, parasites and rotavirus) in a subsample showed no differences between both groups (G2:38.7%, 23.4%, 9.1%; G1: 46.3%, 32.4% and 5.7%, respectively). The species detected were comparable to those of other studies in the same area and the same age group. There were no differences in parameters of nutritional status throughout the study and between the groups. In conclusion, the formula evaluated was associated with a decrease in incidence and a lesser number of days with acute diarrhea.

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