

In a group of 15 patients with hiperinsulinic hypoglycemia we studied the response to Diazoxide treatment. Glucose (O.G.T. T.), Glucagon, leucine and milk have been used as provocative tests for insulin release. The insulin was measured by radioimmunoassay. The hiperinsulinism demonstrated in all patients in response to some of the stimuli was treated with Diazoxide 10mg/Kg/day. After 10 days of treatment the response was evaluated by O.G.T.T. and/or milk test.

Partial or total pancreatectomy was performed in five patients who didn't improve with treatment. Pathologic lesions were as follows: 3 neisidioblastosis, 1 islet cell adenoma and 1 islet cell hyperplasia and hypertrophy.

From our data we concluded:

1° Children who failed to respond to Diazoxide had pathologic lesions in the pancreas.

2° Glucagon seems to be the best stimulus to evaluate this patients.

Furthermore, the milk test has shown to be easy and valuable for evaluation of the therapeutic effect of Diazoxide.

The dopa decarboxylase enzyme (Carbidopa; C) by inhibition of peripheral utilization of L-Dopa (L.D) increases the amount of L.D available to the brain and therefore augments the GH response. This association has been postulated as another test for GH secretion. Fifty one patients with short stature of non-endocrine etiology were studied. In all of them GH secretion was studied under Insulin stimulation (I; 0.05U/Kg) and consecutively to 25 of them under L.D (125-500mg/Kg). In 12 of the 51 the L.D was decreased to 1/2.5 of the initial dosage combined with carbidopa (L.D 1/2.5 + C; 50-200mg/Kg) and in 14 patients the L.D was decreased to 1/5 of the initial dosage plus C (L.D 1/5 + C; 25-100mg/Kg). GH secretion was compared under the different tests.

Results	BASAL		MAXIMAL RESPONSE		GH ng/ml
	GH ng/ml	I	L.D	L.D 1/2.5+C	L.D 1/5 + C
n	51	51	25	12	14
\bar{X}	3.63	13.74	9.32	11.12	8.71
SD \pm	4.03	8.96	5.20	10.65	4.41

I vs L.D: $p < 0.01$ I vs L.D 1/2.5+C: $p < 0.5$; N.S. I vs L.D 1/5+C: $p < 0.005$. Conclusion: The GH secretion under L.D 1/2.5+C is comparable with the I test. The absence of side effects (nausea, emesis) makes this test preferable to L.D alone.

CF is one of the most important causes of Ps.a. lung infection in children, and there is no much knowledge about the mechanisms involved in the lung clearance of this microorganism. The aim of this study was to evaluate the Ps.a. lung clearance in mice with cribriform degeneration, which have been indicated as a possible animal model for CF. Male and female 15-150 days old mice of the DBA/2J-cri inbred strain, genotypes cri/cri and +/? were submitted to an infective aerosol containing Ps.a. Half of the animals were killed immediately and the others 4 hs. after exposure, the lungs were excised, homogenized and cultured quantitatively. Correlation between Ps.a. lung clearance and age was observed as well as an overlapping of the values from both genotypes in mice of the same age. Furthermore, the young animals appear to be more susceptible to Ps.a. infection before weaning than the adult do. The results suggest the existence of a slower maturation of a mechanism involved in the lung defense against Ps.a. infection.

We have measured the IgE levels in 57 patients, aged 4 m. to 6y. with familial or personal history of allergies and recidivant respiratory infections without symptoms or signs of asthma. (group 1). We also studied 110 patients aged 5m. to 14y. with asthma, without other allergic diseases (group 2) and 23 patients aged 6m. to 15y. with asthma and other allergic conditions associated (eczema) and compared the values thus obtained. The IgE levels were measured by RIA Prist method and the results expressed as U/ml. The levels of IgE obtained were compared with the reported values of Kjellman et al. in 125 normal children. In group 1 50,9 % of the patients have IgE levels higher than 2 S.D. above the normal mean ($\bar{X} + 2$ S.D.); group 2 have IgE levels higher than $\bar{X} + 2$ SD in 78,2 % and group 3 have IgE levels higher than $\bar{X} + 2$ SD in 91,3 %. It is clear that IgE levels increased in relation to an augmented atopic charge. We think it is of prognostic interest to detect augmented IgE levels in children with recidivant respiratory infections.

Laboratory of Virology-Serology. "R.Gutierrez" Children's Htal. Buenos Aires. Argentina.

We detected E. COLI K1 capsular antigen by counterimmunoelectrophoresis (CIE) in CSF from 14 neonates with clinical features suspected of meningitis. The mean values of CSF findings were: albumin, 39.3mg%; glucose, 66.5mg%; and 4.5 lymphocytes/mm³. No patient was previously treated with antibiotics, but all of them received them after the first lumbar tap. The illness had a favorable evolution and clinical symptomatology improved within 48hs. CSF cultures did yield no pathogens. We found E.COLI K1 antigen by CIE in 2 cases in feces and in 100-fold concentrated urine of 2 patients without urinary infection. Coagglutination test was positive in 2 CSF samples. Stool cultures yielded E.COLI in 4 cases. Six additional neonates were studied: 4 cases of meningitis and 2 of pyoventriculitis. CIE for E.COLI K1 was positive in the CSF of all of them. E.COLI K1 antigen detected from neonates without meningitis was interpreted as depending on CSF contamination by capsular antigen (destroyed by host immunologic mechanisms) passing from blood stream.

An antimeningococque vaccine prepared with polysaccharide C of Nesseria meningitidis was applied to volunteers from the staff of Hospital de Niños, Buenos Aires. Out of a total of 115 persons, 59 were vaccinated and the other 56 received placebo. The cases were matched by professional activity (nurses, resident doctors, staff doctors and other personnel) and by more or less direct contact with children with meningococcal diseases. The evaluation of the vaccine was carried out by the study of antipolysaccharide C antibodies (passive hemagglutination). One month later 73.2% of vaccinated had increased the antibodies in 2 or more dilutions (66.1% in 3 or more). Only 4% of controls had increased antibodies in 2 dilutions. The Geometric Mean (GM) of vaccinated was 5.5 before and 33.5 one month after vaccination. The GM increased only from 5.35 to 6.02 in controls. One year after vaccine, the GM of vaccinated was 31.34 and even 3 years later there were a significant difference between vaccinated and controls (GM 22.01 and 10.2). Neither professional activity nor the contact with sick children showed differences in the evolution of antibodies.