35

ACTH STIMULATION TESTS IN GIRLS WITH PREMATURE PUBARCHE.
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Premature pubarche (PP) may be due to mild errors of adrenal steroidogenesis due mainly to a deficiency of 21-hydroxylase. However, there is controversy regarding whether all children should undergo an ACTH test in order to differentiate nonclassical congenital adrenal hyperplasia (NCAH) from other causes of PP. An ACTH stimulation test (0.25 mg/IV) with blood sampling both basally and at 60 min was performed in 29 girls with PP. Four patients had acne, 3 had clitoral enlargement and 2 had advanced bone age. Their chronological age was 5.7 ± 1.3 yr, their bone age was 7.7 ± 1.3 yr with an advance of 0.8 ± 0.7 yr, and the height age was 7.3 ± 1.3 yr. Cortisol was normal with elevated baseline levels of 17OHP (69%), ANDRO (68%), DHEAS (64%) and TESTO (21%). The patients were subdivided into 3 groups (G) based on their levels of 17OHP during the ACTH test.
· G1: normal (< 300 ng/dl), n=20; 181.2±76.6 ng/dl; · G2: indeterminate (≥ 300 and < 1000 ng/dl), n=7; 435.8 ± 98.4 ng/dl;
· G3: NCHA (> 1000 ng/dl), n=2; 3810.7 ± 3910.1 ng/dl. There was a statistical difference between 17OHP at 60 min in G1 and G2, ($p < 0.0001$). The proportion baseline value > 100 ng/dl among the 3 groups was different ($p < 0.005$). Clinical findings and bone maturation do not differentiate these groups. We suggest that an ACTH test should be performed only when the 17OHP basal levels exceed 100 ng/dl. In these cases the ACTH stimulation test allows the correct classification of these patients.

36

EVALUATION OF CORTISOL SECRETION WITH THE INSULIN TOLERANCE TEST IN SHORT NORMAL CHILDREN. Bergadá, I., Ropelato, M. G., Heinrich. J. J., Bergadá, C. Division of Endocrinology, Ricardo Gutiérrez Children's Hospital, Buenos Aires, Argentina.
The insulin tolerance test (ITT) is a classic method to assess the secretion of growth hormone (GH) and cortisol. The objective of this study was to determine the dynamics of cortisol secretion after hypoglycemia in 31 normal children (F=9, M=22) with short stature, normal growth velocity and normal GH response to ITT and/or clonidine. Mean chronological age was 12.4 ± 2.7 years. Glucose, cortisol and GH during ITT (0.1 IU/kg i.v.) were measured at -20,0,30,60 and 90 minutes. All the patients developed significant hypoglycemia with a decrease in blood glucose from 79.8 ± 8.9 to 34.1 ± 9.8 mg/dl. We found a significant inverse correlation between basal cortisol and peak increment of cortisol, ($r=-0.79$, $p<0.0001$). Patients were divided into two groups according to the level of basal normal cortisol for age and sex. Group A (n=13): basal cortisol levels within the normal range; Group B (n=18): basal cortisol above 2 SD from the normal mean levels. In group A we found a significant increment in cortisol during the ITT (ANOVA, $p<0.0001$), while group B did not show a significant increment in cortisol during the test in spite of having similar hypoglycemia as Group A (ANOVA). There were no differences in peak GH during the test between the groups.
In summary, we found that the basal level of cortisol influences significantly its increment during ITT, the greater the basal levels, the lower the increment. We believe that this finding, as well as the documentation of the degree of hypoglycemia, is important to interpret the results of an insulin tolerance test.