63 ULTRASONOGRAPHY IN DIFFUSE THYROID DISEASES IN CHILDREN.

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The echo patterns of diffuse thyroid lesions in children are not yet well known. We present here the ultrasound findings of 32 children aged 5 to 15 years with a newly diagnosed diffuse thyroid disease. 23 patients had thyromegaly by palpation, 9 had hypothyroidism but no goiter. Of the 23 goitrous patients 9 were hypo-, 3 hyper- and 11 euthyroid. The 9 with hypothyroidism had all autoimmune thyroiditis as judged by antithyroid antibodies, and confirmed cytologically in 5. They had all 9 a hypoechogenic patchy, partly nodular thyroid by ultrasound.2 of the 3 with Graves disease had initially a similar pattern than in thyroiditis, the 3rd showed hypoechogenity later. Of the 11 euthyroid patients 8 had antithyroid antibodies and the ultrasound suggested thyroiditis in 5 of them, 3 were judged normal (2 and 1 became later hypothyroid). Of the 3 patients with euthyroid goiter but without antithyroid antibodies the ultrasound finding was normal in 1, showed multicystic thyroid in 1 and suggested thyroiditis in 1 who later had antithyroid antibodies. Of the 9 hypothyroid patients without thyromegaly one had an unusually small but otherwise normal thyroid by ultrasound. The others had an echopoor thyroid, but the patchy and nodular pattern was often less marked than in goitrous hypothyroidism. We conclude that autoimmune thyroiditis gives a typical ultrasound pattern, and ultrasonography gives useful information especially of euthyroid goiter and nongoitrous hypothyroidism.

64 PERICARDIAL EFFUSION IN CONGENITAL HYPOTHYROID INFANTS:AN AETIOLOGICAL CORRELATION.

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Ten children positive at the newborn screening program for congenital hypothyroidism were studied. Confirming diagnosis was made in 8/10 infants,while 2/10 were false positive. Moreover we have considered in the study a patient clinically diagnosed at 19 months (he did not undergo the screening program),in which was demonstrated an ectopic gland.A complete thyroid function evaluation (FT3,FT4,T3,T4,TBG,TRH test),scintigraphy (TC99),echocardiography (B-mode)were carried out in all the babies (scintigraphy was not performed in the 2 false positive children). Pericardial effusion was demonstrated in 4/9 patients (44%); complete resolution occurred after a variable period of L-T4 therapy (15 days to 5 months).None of the patients showed clinical symptoms nor electrocardiographic signs of cardiac failure. Based on the aetiology the distribution was the following:

	false positives	ectopic gland	agenesis - dyshormonogenesis	
pericardial effusion	0/2	0/5	4/4	X^2= 5.4 p 0.025

Pericardial effusion correlates with the aetiology and probably depends on the severity of the thyroid failure. The high incidence of this finding in congenital hypothyroid children at diagnosis suggests the need for an initial L-T4 dosage lower than the dosage usually recommended (in our patients 5 to 7 mcg/kg/day allowed the normalization of FT3 and FT4 levels within 15 days of treatment) to avoid cardiac disturbances.

65 NEONATAL HYPOTHYROIDISM IN ENDEMIC GOITER IN ALGERIA.

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Data collected in Zaire suggested that severe iodine deficiency resulted in neonatal hypothyroidism and its longterm consequence, endemic cretinism. In order to further evaluate this possibility, we initiated neonatal thyroid screening in an endemic goiter area in Algeria (area A) with a high prevalence of goiter (51.3 %) and cretinism (1.1 %) and a low dietary supply of iodine (I)(Mean urinary I : 16.2 µg/g creatinine) and in a non goitrous area (B) with no cretinism and a normal iodine supply (urinary I:73.9 µg/g creat.). Serum TSH levels in 3135 newborn infants of area A were shifted towards high values as compared to the results obtained in area B (n=3136). Consequently, for a similar cut-off point for TSH of 40 µU/ml serum, the recall rate was higher in area A (14/3135=0.45 %) than in area B (1/3136 = 0.03 %). The control examinations in the 15 infants recalled were normal in 8 but confirmed severe hypothyroidism in the last 7 infants, all from area A (mean serum TSH:187 µU/ml, T4:2.7 µg/dl). They were put on T4 therapy. Follow up and reevaluation of the diagnosis at the age of 18 months (5/7 infants) including thyroid scintigrams and withdrawal of therapy evidenced thyroids in normal position in the 5 infants, transient hypothyroidism in 3 and persistent hypo.in the last 2 infants. In conclusion, the incidence of neonatal hypothyroidism is elevated in endemic goiter in Algeria (1/450 newborns). Hypothyroidism is transient in most infants but possibly permanent in others. This situation could explain the presence of mental retardation and cretinism observed in adults in endemic goiter in Algeria.

66 INAPPROPRIATE TSH SYNDROME (ITSHS)-TRIIODOTHYRONINE (T3) THERAPY.

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Therapy of ITSHS, not secondary to a pituitary tumor, has proven difficult and has rarely been completely successful. We report a six yr old with mild symptoms of hyperthyroidism and non-tumorous ITSHS who subjectively and chemically partially responded to exogenous T3. A TRF test showed:

	T4 µg	T3 ng	TSH µU	PROLACTIN ng/ml
BASELINE	20.4	450	3.1	13.1
30'	19.8	364	16.1	39
60'	19.8	478	14.4	22
180'	17.6	547	3.6	10

I-131 uptake was 88 and 86% at 4 and 24 hrs; T3 was administered at 25 µg b.i.d., his clinical symptoms subjectively improved, the T3 was slowly increased to 100 µg/day; propranalol was added after a cardiac arrhymia during surgery. A repeat TRF test after 18 months of treatment showed:

	T4 µg	T3 ng	TSH µU	PROLACTIN (PROL)
BASELINE	22	503	1.9	4.1
30'			6.0	12.5
60'	24.9	718	4.9	7.6
180'			2.5	2.2

Basal T4 remains very high, but the TSH and PROL response are more appropriate. I-131 uptake was 24 and 43% at 4/24 hours. The results are compatible with a generalized resistance of tissues to thyroid hormones. Alternative therapies remain a consideration.

67 The Role of Thyrotropin-releasing Hormone(TRH) and Histidyl-proline Diketopiperazine(HPD) in the Maturation of Thermogenesis in Growing Rats.

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Effects of TRH and its metabolite, histidyl-proline diketopiperazine(HPD) on the maturation of thermogenesis were examined. Week old Wistar rats were administered 0.04, 0.4 or 4 nmoles of TRH or HPD intrathecally for 7 days. Half of the litters served as saline-treated controls. Body temperature upon cold exposure (5°C, 1-3 hrs) was measured weekly. On the 4th week of age, catecholamines in forebrain, midbrain, cerebellum, brain stem and adrenal glands were measured by HPLC with an EC detector. NADPH-dependent cytochrome C reductase(CCR) and ATPase activities were determined in liver mitochondria and microsomes. TRH showed a dose-dependent thermogenic effect while HPD potentiated hypothermia. This was associated with an elevated or reduced mitochondrial CCR activity in the TRH and HPD groups respectively. No significant differences were noted in either mitochondrial ATPase or microsomal CCR activity. Norepinephrine(NE) and dopamine in midbrain, cerebellum and brain stem were decreased in both groups but adrenal NE was increased in the HPD group. Dose-related changes were not apparent at 4 weeks of age. These results indicate that TRH and HPD exert antagonistic effects on thermogenesis and mitochondrial NADPH-dependent CCR activity and that these changes may be mediated by central and adrenal catecholamine release.

68 ONTOGENETIC DEVELOPMENT OF THYROTROPIN-RELEASING HORMONE (TRH) IN HUMAN PANCREAS

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Pancreas is the most relevant extrahypothalamic source of TRH. We previously reported that pancreatic TRH content in rats is higher at birth and declines after few weeks. In the present study the ontogenesis of pancreatic TRH in human fetuses and in infants has been evaluated. 12 fetuses of 15-36 w of gestation were obtained after pregnancy interruption; their pancreata and those of 2 infants died at 1 yr were taken within 8 hr. TRH was measured by a RIA in methanolic extracts of whole pancreas and the results were expressed as pg/mg of wet tissue weight. TRH was detectable at 15-21 w of gestation with values from 0.5 to 1.5 pg/mg; a progressive increase was observed with a maximal value of 6.4 pg/mg at 28 w, while a progressive reduction was observed up to the birth, when the mean value was 2.4 pg/mg; in infants of 1 yr the value was 1.5 pg/mg. Human pancreatic TRH had chromatographic, immunoreactive and serum inactivation properties similar to synthetic TRH. The rise of pancreatic TRH content from 15 to 28 w was paralleled by a similar increase of pancreatic content of insulin. In conclusion, the TRH is present in human fetal pancreas and its ontogeny is characterized by a biphasic pattern.