47 TSH METABOLISM IN THE NEWBORN LAMB. J. Sack and D.A. Fisher, Dept. of Pediatrics, UCLA-Harbor General Hosp. Torrance, California, U.S.A.

We have reported that serum T3 in the lamb increases in the newborn period and that this increase seems to be provoked by cutting the umbilical cord (CCT). To further clarify the role of TSH in the newborn lamb we have studied serum OTSH levels and the response to exogenous TRH.

In controls serum TSH levels increased modestly to peak levels (mean 215%) at 30 min. A similar 30-45 min. peak (mean 160%) occurred in newborns in which CCT is delayed 60 min. In spite of the early OTSH Δ the T3 response was delayed until after CCT. The response to TRH (230 µg injected intravenously) was studied in 8 lambs. Four were under 6 hrs. of age and 4 were between 7-18 hr. Blood was sampled serially for measurements of TSH, prolactin, T4 and T3. There was a rapid rise in serum TSH and prolactin conc. in the first 15 min.: from a mean of 7.3 µU/ml and 571 ng/ml at 0 time to 14.2 µU/ml and 2130 ng/ml, respectively. A second peak was observed at 120 min. after TRH, both for TSH (24.6 µU/ml) and prolactin (2149 ng/ml), suggesting new synthesis and release. T4 and T3 levels began to rise between 60 and 120 min. Peak levels were achieved 6 hr. after TRH: T4 Δ 12.6 to 22.2 µg/100 ml and T3 Δ 221 to 320 ng/100 ml.

These data indicate 1) the TSH response in the newborn lamb is much less marked than in the newborn infant 2) the TSH and T3 responses can be dissociated by CCT 3) the T3 response is not due to TSH Δ 4) the newborn lamb responds to TRH with rapid increases in TSH and prolactin.

48 ALPHA CELL FUNCTION IN SHORT, OBESE, AND DIABETIC CHILDREN. P.Schneider, A.Rabinovitch, L. Paunier and P.C.Sizonenko, Department of Pediatrics and Genetics, and Institute of Clinical Biochemistry; University of Geneva, Geneva, Switzerland.

Plasma glucagon (IRG) and insulin (IRI) responses to intravenous infusion of arginine (A:0-60 min) plus glucose (A+G:30-60 min) were examined in 7 children with idiopathic short stature (SS), 6 with moderate obesity (ob), 7 with gross obesity (OB), and 3 juvenile diabetics (DB). Mean (±SE) plasma IRG (pg/ml) was highest in the OB group :

	Basal	A	A+G	post A+G
OB	163±38	299±47	226±29	181±57
ob	52±20	138±34	81±28	66±12
SS	67±15	137±35	106±23	63±13
DB	109±34	252±91	346±100	79±31

Similar fold (x2-3) increases of plasma IRG were seen for all groups during A, as well as similar decreases of IRG during A+G. IRI responses to A as well as A+G were smaller in the SS group. These data suggest that plasma glucagon responses are intact and may even be exaggerated in obesity.

49 SIMULTANEOUS DETERMINATION OF SIX PLASMA CORTICOSTEROIDS IN PUPPIES DURING STANDARDIZED HAEMORRHAGIC SHOCK. W.G. Sippell, W. Golder, H.Hahn, P. Lehmann and G. Hollmann (Intr. by H. Helge), Experimental Surgical Laboratory, University of Munich, Children's Hospital, München, Germany

11 puppies of 2-3 weeks of age and either sex were subjected to a standardized haemorrhagic shock. Aldosterone (A), corticosterone (B), cortisol (F), deoxycorticosterone (DOC), progesterone (P) and 17α-OH-progesterone (17α) were determined simultaneously in 250 ul of peripheral plasma taken at constant dates before, during and after blood withdrawal and re-infusion. Our method included CH_2Cl_2 extraction, separation by automated Sephadex LH-20 multi-column chromatography and quantitation of each steroid by radioimmunoassay.

Steroid (ng/ml)	Control Period	Hypovolaemic Shock Phase	normovol. Phase 1h	2h	4h
A	0.5	3.2	2.7	1.8	2.4	2.5
B	22	47	51	31	43	56
F	72	152	164	177	226	187
DOC	0	1.2	2.7	0	0.4	0
P	0.9	4.4	8.4	0	2.4	0
17a	5.1	8.0	6.3	2.2	0.02	0.05

During hypovolaemia, plasma levels of the main mineralocorticoid (A) and its precursors (P,DOC,B) increase much more than those of the main glucocorticoid (F) and its precursor (17α). These and additional data on electrolyte and glucose levels reflect the key role of the adrenal cortex in maintaining homoeostasis during shock in the early life period.

50 SUBNORMAL GROWTH HORMONE (GH) LEVELS WITH NORMAL GROWTH. N.Stahnke, D. Schönberg, P.Steins, R.P.Willig, and W. Blunck, Dept. of Pediat. and II.Dept. of Medicine, University Hamburg; Dept. of Pediat., University Heidelberg, Germany.

4 children with a pituitary or hypothalamic tumor were studied. 3 of them underwent surgery, post-operatively a growth-spurt followed by normal growth as observed. One of these patients showed an excessive weight gain. The fourth patient in whom the tumor was not removed had a normal growth rate as well. In all 4 patients endocrine evaluation revealed varying degrees of anterior and posterior pituitary insufficiency. Peak GH levels following various stimuli were extremely low in all patients. Prolactin activity in serum as measured by a local pigeon crop assay was elevated in one patient only. In all patients the serum activity of somatomedin was determined by bioassay, serum levels of insulin were measured by RIA during stimulation tests. Our findings suggest that a biological substance other than GH might have induced somatomedin generation in these patients.
Supported by Deutsche Forschungsgemeinschaft, SFB 34

51 REMARKABLE CATCH-UP GROWTH IN A BOY WITH THE NEPHROTIC SYNDROME, TREATED WITH PREDNISONE. R.Steendijk, Department of Paediatrics, University of Amsterdam.

In a boy, who got the nephrotic syndrome at the age of 4 years, growth and development were followed for 20 years. During this time glomerular function remained adequate. Initially, height was 101,5 cm (-0.8 SDS). During daily administration of prednisone in a dosage between 0.2 and 0.6 mg/kg/day, growth and development became retarded until - at the age of 15 years - height was 138.0 cm (-3.5 SDS) and S.A. (TANNER and WHITEHOUSE II) was 8 years. From that time prednisone was given every other day or 3 times per week in a dosage which slowly decreased from 2 to 1 mg/kg/week. Growth rate increased gradually from 1 cm/year at the age of 14 years until 8 cm/year at the age of 19 years, whilst the difference between C.A. and S.A. slowly decreased. Puberty began at the age of 17 years. At the age of 24 years height was 171.5 cm (-1.0 SDS) and S.A. was 18.0 years. Noteworthy features were: 1) Anachronism between the RUS- and CB-scores for S.A. amounting to 1.6 - 3.0 years during most of the time; and 2) a predicted adult height (TANNER et al., 1975) which was too low, the difference with achieved adult height being larger with increasing discrepancy between C.A. and S.A.

52 TESTICULAR ENDOCRINE FUNCTION IN PATIENTS TREATED WITH CYCLOPHOSPHAMIDE FOR NEPHROTIC SYNDROME. H. Stolecke, H. Olbing and H.-J.Bachmann, Children's Hospital, Department of Endocrinology and Nephrology, Univ. Essen, FRG.

Reports of damaging effect of cyclophosphamide on the germinal epithelium led us to study as whether the endocrine function may be also involved in cyclophosphamide induced side effects. 5 boys aged 9-14 years suffering from nephrotic syndrome unresponsive to steroid therapy were treated for 6-9 weeks with cyclophosphamide 2.5-3.0 mg/kg. Immediately after the end of the drugs period and a second time 13 till 28 months later they were examined by a stimulation test with HCG, using a single injection of 5000 IE/m^2 body surface i.m. Basal and 1, 3 and 5 days after HCG 24 hrs urine samples as well as plasma samples (at 9 a.m.) were analyzed for pregnanetriol (P3OH) and for testosterone respectively. P3OH was determined by GLC, T by RIA.

In the 2nd series urinary P3OH increment related to bone age was sufficient in 4 patients, but in 3 cases with a tendency to decrease while compared with the values immediately after the drugs period. Plasma T-values were normal under basal conditions. After HCG 2 patients increased their T-values as so far to be expected for a normal reaction, the 3 others showed a sluggish response only. From our results we suppose that cyclophosphamide therapy may involve a risk of a damaging effect also on the endocrine part of testicular function.