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Serum concentrations of TSH,  $T_4$  and  $T_3$  in fullterm, preterm and small-for-gestational age newborns. A total number of 103 fullterm (FT), 47 small-for-gestational age (SGA) and 35 preterm (PT) babies were studied from day 1-8 after birth. In FT a surge of TSH was invariably seen, followed by a decrease until normal adult levels were reached on day 2-4 after birth. Serum  $T_4$  and  $T_3$  concentrations were higher than in adults, significantly correlated and decreased by 30 and 40% respectively from day 2 to 8 after delivery. In SGA the postnatal TSH hypersecretion was more variable. Serum  $T_4$  and  $T_3$  concentrations were significantly correlated and lower than in FT. Serum  $T_4$  decreased insignificantly and serum  $T_3$  seemed to increase during the first week of life. In PT the postnatal TSH increase was highly variable. Serum  $T_4$  and  $T_3$  were lower than in FT and did not change significantly during the study period; serum  $T_3$  tended to increase. Birth weight as well as gestational age were correlated with the thyroid hormone concentrations. In babies with gestational age of more than 34-36 weeks normal serum  $T_4$  and  $T_3$  concentrations were often observed. It is concluded that the postnatal pituitary-thyroid hyperfunction may be subnormal in SGA and PT babies. If serum TSH concentration is not measured in screening for congenital hypothyroidism these babies may appear as false positives.

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Pituitary LH,  $\alpha$ - and  $\beta$ -subunits during perinatal life. In order to evaluate pituitary LH secretion, total LH (including hCG) and its  $\alpha$ - and  $\beta$ -subunits were measured from birth to the 7th day of life by radio-immunoassays. Since no sex difference was observed, data will include both sexes. In 6 male and 6 female newborn infants, "LH-hCG" decreased from  $38.2 \pm 5.7$  mU/ml (MRC 69/104) at birth to  $8.1 \pm 0.7$  ( $p < 0.01$ ) at days 3-4 and increased to  $12.7 \pm 1.6$  at days 6-7. In cord blood, specific plasma LH levels (Lequin's anti-LH $\beta$ ) were  $6.3 \pm 0.2$  ng/ml (LER 960) in both sexes. Plasma LH decreased to  $4.3 \pm 0.2$  ng/ml on days 3-4 and  $4.4 \pm 0.4$  on days 6-7. In contrast to the Lequin's anti-LH $\beta$ , mean plasma LH with the NIH anti-LH $\beta$  was lower in cord blood ( $1.6 \pm 0.3$  ng/ml). Mean level of  $\alpha$ -subunits (LH- $\alpha$  radio-immunoassay) was  $55.9 \pm 7.2$  ng/ml in cord plasmas. Alpha-subunit levels markedly decreased to  $3.8 \pm 0.8$  ng/ml on day 1-2 and remained at  $4.1 \pm 0.3$  ng/ml between days 3-7 in both sexes. In conclusion, hCG decreases very rapidly after birth; hCG $\alpha$  represents the bulk of  $\alpha$ -subunit in cord blood. Pituitary LH levels are low during the first 5 days of life.

**54** P. CZERNICHOW, A. BASMACIOGULLARI, P. TOUBAS\* Y. DUMEZ\* and C. TCHOBROUTSKY\*. Hôpital des Enfants-Malades and Hôpital Port Royal\* Paris, Fr. Arginine-Vasopressin (AVP) secretion and placental transfer in maternal (M) and foetal (F) sheep.

AVP secretion and placental transfer were studied from 126 to 137 days' gestation in chronically cannulated F and M sheep. Two types of experiments were performed during which blood was collected simultaneously in M and F and AVP measured by radio-immunoassay.

1) Hypotensive hemorrhage (15% blood volume) were performed in M or in F. An acute secretion of AVP was observed during hypotension. Values (pg/ml and range) are as follow.

		n	base	peak
Hemorrhage	M	5	2.1 (1-3)	169 (44-560)
	F	5	3.6 (2-4.5)	10.4 (4-24)
Hemorrhage	F	1	3.4	24.5
	M	1	4.5	6.2

2) Synthetic AVP was injected in the M or F circulation (n=2). No significant transfer was found in either direction. AVP half life in the F was 6 and 5 min with a distribution volume of 0.67 l and 0.32 l. Plasma half life could not be calculated in the M compartment. In conclusion 1) plasma AVP was found in the F, 2) preliminary experiment indicated that the F posterior pituitary was stimulated by foetal HH, 3) there was no evidence for placental transfer of AVP.

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Umbilical cord cutting: the stimulus to the glucagon surge in newborn lambs.

A prompt increase in glucagon (GLN) attributed to the fall in glucose (G) after birth has been observed in rat and human newborns immediately after delivery. This GLN surge also occurs in the newborn lamb, but is not preceded by a fall in G. To further explore this phenomenon, we investigated the term lamb during 1) delivery (D) and immediate umbilical cord cutting (CCT); 2) D with CCT delayed for 60 min. and 3) infusion of cyclic somatostatin (SRIF) to the fetus in utero and for 60 min. post D, while CCT was performed immediately after delivery. All animals were delivered by Caesarean section under local anesthesia. The characteristic surge in GLN was identified in 1, but did not occur in 2 while the cord was intact. However, after CCT in 2, plasma GLN rose from  $59 \pm 15$  pg/ml (mean  $\pm$  SEM) immediately prior to CCT, to  $304 \pm 99$  pg/ml 15 min. later ( $p < 0.05$ ). G did not change, confirming that hypoglycemia was not the stimulus for GLN secretion. Intravenous SRIF (50  $\mu$ g bolus followed by 200  $\mu$ g per hr) begun in the fetus 10 min. before delivery did not affect low fetal GLN. Despite ongoing SRIF, CCT was followed 15 min. later by a surge in GLN ( $\Delta$  130 pg/ml;  $p < 0.01$ ) and at 30 min. by a rise in G of 38 mg/dl ( $p < 0.05$ ).

Conclusions: a) CCT evokes the neonatal GLN surge, b) the GLN rise is not related to curtailment of G supply, and c) unlike other stimuli for GLN, umbilical CCT overrides the suppressive effects of SRIF by undefined mechanisms.

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Growth in dialysed and transplanted children.

The European Dialysis and Transplant Association (E.D.T.A.) conducts a yearly survey on growth in children undergoing regular haemodialysis (HD) and renal transplantation (TR). According to the results obtained at the end of 1974 body height of 474 children starting a kidney function replacement programme was below the 3rd percentile (P.) in 40%. Growth retardation was more severe in children with congenital than with acquired kidney disorders. In boys (up to age 16) and girls (up to age 14) growth velocity (GV) was  $<$  3rd P. in 70% of patients on HD and in 63% after TR. In older HD patients GV was  $<$  3rd P. in only 13%. The pubertal growth spurt seems to be delayed and depressed in children on HD. In boys followed for 1-2 years on HD GV was 3.0 cm/y. vs. only 2.3 cm/y. in those followed for  $>$  3 years. With more frequent and longer dialyses per week GV was better in males, but not in females. In order to obtain better insight in the factors leading to growth failure on HD a more detailed survey was undertaken at the end of 1975, including data on bone age, nutrition and pubertal status.

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Aetiology of Idiopathic Growth Hormone Deficiency in England and Wales.

The relative importance of heredity, sex and the conditions of pregnancy in the aetiology of idiopathic GH deficiency was investigated in a case-control study of 140 patients. Certain items were investigated in a further 68 patients. In the main sample the ratio of boys to girls was approximately 4 to 1. Half of the affected children were first-born which is a significantly different proportion from the control population ( $\chi^2_1 = 6.77$ ;  $p < 0.01$ ). The affected children had an incidence of breech delivery (11.4%) that was very significantly increased compared to controls ( $\chi^2_1 = 53.2$ ,  $p < 0.001$ ). They also presented a significantly high incidence of forceps delivery (12%;  $\chi^2_1 = 16.12$ ,  $p < 0.001$ ). Breech and forceps delivery considered together occurred significantly more frequently in cases with multiple pituitary deficiency (43.1%) than in those with isolated GH deficiency (20%;  $\chi^2_1 = 3.92$ ,  $p < 0.05$ ). Early vaginal bleeding during pregnancy was significantly increased (7.5%;  $\chi^2_1 = 7.05$ ;  $p < 0.01$ ). The seasonal distribution and parental ages were normal. The true probability of a proband having another affected sib was  $2.9 \pm 1.5\%$ . The relative risk of GH deficiency in males born by breech in primiparae is 10.5 times that of the general population; that of females, vertex in multiparae, 0.2 times. Routine screening of the population at risk is proposed.