11 Physiologic GH secretion evaluation by means of mean nocturnal GH concentration assessment. G.L. Spadoni, S.Cianfarani,S.Bernardini,B.Boscherini.Dept. of Peadiatrics, IInd University of Rome.

Standard provocative stimulation tests for GH cannot always determine the actual physiologic secretion of GH.Subjects have been described who respond to pharmacologic tests but show a reduced physiologic secretion as evaluated by means of multiple serum GH measurements over 24 hours and successfully respond to hGH therapy.Reliability of a 12-hr GH spontaneous secretion evaluation was assessed.27 subjects with short stature due to familial short stature (FSS),constitutional growth delay (CGD),total GH deficiency (TGHD),partial GH deficiency (PGHD) or with idiopathic short stature (ISS) were studied. Mean nocturnal GH concentration (MNGHC)shows a statistically significant difference between controls (children with FSS) and children with TGHD (P<0.001)with no overlap between the two groups.MNGHC shows intermediate values in children with PGHD.All the patients with ISS,who had a normal response to pharmacologic tests,showed a reduced MNGHC.One of these patients showed a good height velocity increase during hGH therapy.In all subjects a significant correlation (P<0.001) between MNGHC and plasma SMC levels was observed while no correlation resulted between MNGHC and maximal GH response to pharmacologic stimuli. A significant correlation (P<0.001) between MNGHC and height velocity was observed. Our data show the full reliability of physiologic GH secretion assessment over a 12-hour night-time interval.

12 Comparison between lung glucocorticoid receptor concentrations in fetus, infant and children.

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The role of glucocorticoid in prenatal lung maturity is all important.
They act on type II alveolar cells through the intermediary of glucocorticoid receptor (GR). The evolution and function of the GR in post natal lung growth are unknown.
We mesure the lung GR in fetus, infants and children at ages corresponding to different stages of alveolar growth.
Human lung tissue was obtained from 9 surgically aborted fetus or stillborn fetus (15 to 28 weeks of gestationnal ages) and from 6 infants and childrens (2 months to 9 years) after lobar resection or at autopsy.
Rates of GR varied 35 to 300 fmol/mg prot in lung fetus (Kd : 7.7±0.7 nM) and 1 to 30 fmol/mg prot (kd : 10.4±2.9 nM) in infant. The comparison between mean concentrations of GR in fetus (182±87 fmol/mg prot) and in infants or childrens (14.6±9.9 fmol/mg prot) show a significant difference ($p < 0.01$).
The values we recorded in infants and children are litle different from GR concentrations reported in adult human lung.
The similarity between these results in comparison with values in fetus would suggest that GR have little effects on post natal lung growth and/or maturation.

13 Optimal prescribing of red cell transfusion in preterm infants.

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Today's tiny babies need frequent red cell transfusions (Tx). If inadequate red cells are Tx on each occasion, the donors and so infective hazards, are multiplied. The objective of Tx is to achieve normal circulating red cell mass (RCM), but to avoid high blood viscosity. Calculations of the volume to Tx based only on the desired haematocrit (Hct) may underestimate the deficit in RCM because of variations in plasma volume.[1] We have studied preterm infants before and 12 hours after Tx for blood loss and refractory anaemias: Group 1 - 13 Tx in babies aged birth - 10 days (mean = 4 days); Group 2 =21 Tx in babies aged 13-159 days (mean 36 days). We compared rises in Hct and RCM achieved with the various volumes of red cells transfused (mean ± 1SD)

Table	Hct Pre Tx	Hct Post Tx	Red cells Tx	RCM achieved
Group 1	0.40 ± 0.10 (0.25 - 0.59)	0.49 ± 0.12 (0.31 - 0.65)	12.4 ± 3.8 (8.4 - 21.0)	36.3 ± 9.9 (20.3 - 55.0)
Group 2	0.29 ± 0.05 (0.20 - 0.36)	0.48 ± 0.08 (0.32 - 0.63)	16.9 ± 5.4 (8.0 - 27.4)	35.0 ± 10.0 (21.4 - 56.6)

We have correlated pre-Tx Hct, volume of red cells* Tx and RCM achieved. Mathematical expressions have been derived for prediction of the volume of red cells* to Tx to achieve any desired RCM in the individual infant. eg. For our group 1, 16-29 ml red cells*/kg (mean 23) would achieve RCM of 50 ml/kg, approx normal for the term infant. To avoid inordinate rises in Hct this may have to be given in more than one aliquot, monitoring the Hct at each stage. For group 2, 10-24 ml red cells*/kg (mean 16) would achieve 35 ml/kg RCM.
(*Pure red cells i.e. Hct = 1.0)

[1]Lancet April 86. i 882. Phillips et al.

14 BLOOD VOLUME ESTIMATION IN NEONATES USING 99m-TC-LABELLED ERYTHROCYTES AND EVANS BLUE

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Mixing and transcapillary loss of the plasma label Evans blue was studied in 84 preterm and term neonates and equilibration of both Evans blue and 99m-Tc-labelled red blood cells (99m-Tc-RBC) was investigated in 35 severely sick neonates with informed consent of the parents. Blood samples were taken 5, 10, 20, 30 and 45 min after injection of the indicators. Mixing time of Evans blue was complete within 10 min in 76 out of 84 neonates. Mixing of 99m-Tc-RBC was delayed to 20-30 min in 14 out of 35 infants. Prolongation of mixing time was related to low blood pressure and to low peripheral blood flow (plethysmography). Disappearance rate of Evans blue increased with increasing blood volume and with decreasing pH. The disappearance rate averaged 17±9 %/h in 21 neonates without serious disorder, 24±10 %/h in 48 neonates with RDS ($P < 0.05$) and 28±7 %/h in 15 neonates with septicaemia ($P < 0.001$). The ratio of the body haematocrit (RBC mass/blood volume) to the venous haematocrit averaged 0.85±0.04 in 35 neonates. This ratio tended to decrease in severely sick neonates. We conclude that both plasma and RBC indicators may yield incorrect results when only one label is used or when only one blood sample is taken after injection of the indicators.

15 ESTIMATION OF DEFORMABILITY OF NEONATAL AND ADULT LEUKOCYTES VIA MICROPIPETTE ASPIRATION

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Leukocytes from normal term neonates, neonates with septicaemia and a high percentage of immature neutrophils and leukocytes from healthy adults were studied after separation. Micropipettes with a diameter of 5 µm were used to determine the pressure required to aspirate whole leukocytes. Smaller pipettes with a diameter of 2.7 µm were used to aspirate membrane/cytoplasma tongues from these cells. Lymphocytes and granulocytes required similar aspiration pressures although lymphocytes are smaller than granulocytes. Monocytes have large volumes and required correspondingly high aspiration pressures. Neonatal granulocytes, lymphocytes and monocytes tended to be larger and to require higher aspiration pressures than adult leukocytes. Neonatal bands and juvenile neutrolphils required very high aspiration pressures. At a constant aspiration pressure of 2 cmH_2O, tongue formation in 2.7-µm pipettes was markedly slower for lymphocytes compared to mature granulocytes, with the values for immature granulocytes and monocytes lying between these two extremes. Tongue formation of neonatal and adult leukocytes followed similar patterns. We conclude that neonatal leukocytes increase vessel resistance more than adult cells due to immature leukocytes and the larger leukocyte size.

16 AORTIC BLOOD FLOW VELOCITY IN THE SGA FETUS: RELATION TO HEMATOCRIT.

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Mean blood flow velocity in the fetal abdominal aorta has been assumed to reflect the degree of placental insufficiency. However, only a poor correlation was found between mean blood flow velocity in fetal aorta (mAoBFV) and the degree of fetal growth retardation. We studied the relation of mAoBFV to umbilical hematocrit (Hct) and to placental weights adjusted to gestational age (% of normal mean). mAoBFV was determined by pulsed doppler ultrasound in 23 fetuses with weights below the 10th percentile for gestational age (28-41 wks gestation). Umbilical artery Hct was 0.53 (0.40-0.70 1/1) (mean, range), mAoBFV 27.2 (11-39 cm/s). A highly significant ($p < 0.001$) linear correlation existed for mAoBFV vs. Hct ($y = -64.8x + 61.3$, $r=0.67$) but not for mAoBFV vs. placental weight ($r=0.33$). As an index of hemoglobin (Hb)-flux we calculated the product of mAoBFV x Hb-concentration. This index of Hb-flux did not change with Hct ($r=-0.23$).
Conclusion: Our data suggest that, in addition to placental vascular changes, an increased hematocrit may contribute to low mAoBFV.