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LANGUAGE AND SPEECH DELAY AT 1, 2, AND 5 YEARS OF AGE IN VERY PRETERM AND VERY LOW BIRTHWEIGHT INFANTS

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Results of language screening in a nationwide collaborative study of a virtually complete year cohort of 1338 infants with a gestational age <32 weeks or with a birthweight of <1500 grams were reported. At the age of 1 and 2 years the language items derived from the Van Wiechen neurodevelopmental assessment and at 5 years the VTO-language screening test were used, both validated for the Dutch language. Language delay was recorded in 16%, 23%, and 24% respectively at 1, 2, and 5 years of age (corrected for preterm birth).

In a multiple logistic regression analysis a relationship of language delay at 5 years was found with perinatal risk factors: level of parental education, multiple pregnancy, birthweight, male sex, and neurological status at discharge. Both language delay at 1 year and at 2 years were predictors for delay at the age of 5 (OR 2.2 and OR 2.9). Disability not due to language delay at 5 years of age was related to language delay (OR 3.2). Particularly disabilities of mental development and neuromotor function and to a lesser extent disabilities of visual function and hearing loss were involved. At 5 years 12% is already attending special education. As language delay is a strong predictor of school failure in the coming years a considerable increase of this percentage is expected.

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MATERNAL DIABETES DOES NOT DECREASE GLUCOSE AND GLYCEROL PRODUCTION IN NORMOGLYCAEMIC NEWBORNS.

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Neonatal hypoglycaemia is frequently observed following pregnancies complicated by diabetes. This might be a result of decreased glycogenolysis/gluconeogenesis caused by neonatal hyperinsulinemia. The aim of the study was to investigate the capacity for production of glucose and glycerol, a glucose precursor, in infants of diabetic and healthy mothers.

Subjects and methods: Eight normoglycaemic, fasting, term infants from diabetes pregnancies (IDDM/GDM) and two control infants were studied at a postnatal age of 4-10 h. The glucose (GPR) and glycerol production rates (GlycPR) were studied by use of 6,6-$^2H_2$-glucose and 2-^{13}C-glycerol. The isotopic enrichments and concentrations of glucose (P-gluc) and glycerol (P-glyc) in plasma were analyzed. Isotopic enrichments were measured by gas chromatography/mass spectrometry during periods of steady state and were used for calculation of production rates.

Results:	**GPR**	**GlycPR**	**P-gluc**	**P-glyc**
	mg kg^{-1} min^{-1}	µmol kg^{-1} min^{-1}	mM	µM
IDDM/GDM	5.0±1.4	10.4±4.1	3.0±0.6	450±62
Control	5.0±0.1	12.9±0.0	3.5±0.2	500±71

Conclusion: Normoglycaemic infants of diabetic mothers have capacity for glucose and glycerol production, at rates similar to those in control infants.

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THERMAL BALANCE IN PRETERM INFANTS NURSED IN AN INCUBATOR WITH A RADIATIVE HEAT SOURCE

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Radiative heat loss is a major source of energy loss for preterm infants nursed in convectively heated incubators. Using a radiant hood warmer, we studied the effect of an isolated change in roof temperature (T_{roof},°C), at stable ambient air temperature (T_{amb},°C) and humidity (RH,%), on the thermal balance of 7 preterm infants (gestational age 29 (27-33) weeks, birthweight 1485 (948-2356) grams, postnatal age 8 (0-13) days).

Respiratory water loss (RWL, mg/kg min), oxygen consumption ($\dot{V}O_2$, ml/kg min), transepidermal water loss (TEWL, g/m^2 h), skin blood flow (Q_s,%, intervall A=100%), skin (T_s,°C) and central body (T_c,°C) temperatures were continuously monitored. After an interval without active heating of the incubator roof (interval A), the roof was heated to 33°C (interval B), 36°C (interval C), and finally to 39°C (interval D). Mean values for each interval, including convective (H_{conv},W/m^2 °K) and radiative (H_{rad},W/m^2 °K) heat losses, are given in the table (*= p<0.01, ' =p<0.05, as compared to interval A)

Interval	T_{roof}	T_{amb}	RH	T_s	T_c	Q_s	H_{rad}	H_{conv}	RWL	$\dot{V}O_2$	TEWL
A	31.3	34.1	50	35.8	36.9	100	29.9	4.4	3.6	4.8	8.3
B	34.5*	33.5	51	35.9'	37.0	103	22.2*	6.4	3.7	4.8	7.8
C	36.6*	33.9	50	36.2'	37.1	111'	17.5*	6.1	3.9	4.6	7.7
D	38.6*	34.3	50	36.4'	37.1	115	11.3*	5.6	3.5	4.8	7.8

Conclusion: Warming the incubator roof results in a marked decrease in radiative heat loss, without any significant change in central body temperature, skin blood flow, respiratory water loss, oxygen consumption or transepidermal water loss.

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STATIC RESPIRATORY SYSTEM COMPLIANCE (CRS): AN EARLY INDEX OF PROGNOSIS AND DISEASE SEVERITY IN THE NEWBORN.

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We compared the prognostic value of static Crs measured in the first 24 hr using a single breath airway occlusion technique (Le Souef et al, Am Rev Resp Dis 1984; 129:552-6) with mean FiO_2 in the first 12 hr in 48 ventilated newborn infants of median (range) birthweight 1270 (480-3500) g. In a further 33 infants, Crs measured before surfactant administration in the first 24 hr was compared to junior doctors' visual estimates of respiratory compliance (i) using a visual analogue scale (analogue Crs) and (ii) based on their assessments of tidal volume (Vt Crs).

Prediction of hospital death	Sensitivity	Specificity	
Crs in first 24 hr < 0.6 ml/cm H2O/m	80%	100%	p < 0.0001
Mean FiO_2 in first 12 hr > 60%	80%	81%	p = 0.014

Junior Doctor	Correlation with measured Crs		95% limits of Agreement	
	Analogue Crs	Vt Crs	Analogue Crs	Vt Crs
Registrar	r = 0.283	r = 0.446	-90 % - +116 %	-63 % - +94 %
SHO	r = 0.676	r = 0.443	-60 % - +62 %	-62 % - +95 %
Res Fellow	r = 0.479	r = 0.505	-65 % - +91 %	-60 % - +89 %

Static Crs provides an estimate of respiratory disease severity which may be less distorted by ventilator management than indices based on blood gases, such as FiO_2. Visual estimates of Crs by junior doctors were unreliable. Routine measurement of Crs may therefore be a valuable adjunct to clinical management.

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Low left ventricular output predicts fatal outcome in babies with persistent transitional circulation (PTC)

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30 newborns with clinical evidence of PTC and structurally normal hearts underwent estimation of left ventricular output (LVO) using Doppler echocardiography, shortly after presentation. Results of survivors were compared with non-survivors.

Study Group 14 babies were at term (birth weight 2585-4390 g) and 16 were pre-term (26-36 weeks, 855-4615 g). Age at examination was 1 to 72 hours, median 20 hours. Underlying diagnoses were hyaline membrane disease (12), asphyxia (10), meconium aspiration (2), maternal diabetes (1), hydrops (1) and hypoplastic lungs (1). 3 babies had no obvious cause ("PFC"). 27 babies were ventilated.

Results Values can be compared with 10th-90th centile ranges from healthy newborns in the first 72 hours of life (LV output 166-248 mls/kg/min, LV stroke volume, indexed by body weight (SVI) 1.2-1.9 mls/kg). 12 of the 30 babies died 19-478 hours (median 25 hrs) after the examination. 2 early deaths were due to severe pulmonary interstitial emphysema and not attributable to PTC and are excluded from this analysis:

	LVO(mls/kg/min)		SVI (mls/kg)	
	Range	Mean	Range	Mean
Survivors (n = 18)	136-329	205	0.71-2.30	1.36
Non-survivors (n = 10)	53-245	138	0.33-1.33	0.86
	p < 0.01		p < 0.005	

4 babies had LVO < 100mls/kg/min, and all 4 died. 6/7 babies with SVI < 1ml/kg died.

Conclusion Low LVO and SVI predicted subsequent death in babies with PTC, and is worthy of further prospective evaluation. The close link to outcome is probably because these are both reduced by a reduction in pulmonary venous return and/or myocardial performance and hypovolaemia, all of which are features of severe disease.

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CHARACTERIZATION OF A SP-A-BINDING PROTEIN AT THE CELL MEMBRANE OF TYPE 11 PNEUMOCYTES WITH THE USE OF AUTO-ANTIDIOTYPIC ANTIBODIES. Paul A. Stevens, Ver Meienreis-Sudau, Bernd Rustow. Dept. of Neonatology and Instit. of Pathological and Clinical Biochemistry, Humboldt University, Charite Hospital, Berlin.

Interaction of extracellular lung surfactant-specific protein SP-A with type II pneumocytes is involved in modulating alveolar surfactant metabolism. SP-A enhances uptake and subsequent recycling of surfactant towards lamellar bodies, the secretory organelles of the type II cells. It is also involved in regulating surfactant secretion by these cells.

The nature of the type II cell component(s) interacting with SP-A has been unclear. After immunizing mice with SP-A we developed a panel of anti-SP-A antibodies as well as two monoclonal antibodies (mAbs) against type II pneumocyte cell membrane components (but not SP-A). In vitro immunization with these mAbs produced anti-SP-A antibodies, demonstrating the auto-antiidiotypic nature of the mAbs.

With the use of these mAbs we identified a specific SP-A-binding protein (MW 180-210,000) on type II pneumocyte cell membranes. As shown by 1- and 2-D gel electrophoresis, this protein consists of subunits, MW 55,000. (Biochemical characterization of the protein and its subunits will be presented.) Our results indicate that this protein may be involved in surfactant metabolism regulation.

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