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ETHANE AND PENTANE IN EXPIRED AIR. MEASUREMENT OF LIPID PEROXIDATION IN SMALL PRETERM INFANTS. Olli Pitkänen, Sture Andersson, and Mikko Hallman, Univ. Helsinki, Dept. Pediatrics, Helsinki, Finland

Free oxygen radicals are claimed to be of major importance in tissue damage in the newborn. Ethane (E) and pentane (P) are specific products of free oxygen radical mediated lipid peroxidation (Lipids 12: 109-114, 1977). We have quantified these alkanes in expired air from 12 infants, gestation 25 to 31 weeks. All received mechanical ventilation and supplemental oxygen for treatment of RDS. After wash-out with hydrocarbon scrubbed air/O₂, the expired air was collected into a bag, trapped into Porasil-C adsorbent, and analyzed with internal standard by gas-chromatography.

During the first three neonatal days E production ranged from 0 to 133 pmol/kg/min and P from 0 to 39.5 pmol/kg/min. E and P did not correlate well with the severity of the respiratory failure or with the requirement of supplemental oxygen during the time of gas analysis. However, infants who eventually developed BPD (n=2) or died (n=2), tended to produce more E (63.4 pmol/kg/min, range 0-133), and P (29.1 pmol/kg/min, range 12.9-39.5), compared to babies with a favourable prognosis: 22.9 pmol/kg/min (0-55), and 11.3 pmol/kg/min (0-22), respectively. We propose that analysis of E and P is instrumental in studying the role of free oxygen radicals in diseases of the newborn.

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FLOW BEHAVIOR OF NEONATAL AND ADULT ERYTHROCYTES IN VESSELS WITH DIAMETERS OF 3 TO 6 μ m
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This study was designed to analyse the flow behavior of red blood cells (RBC) by means of a mathematical model. According to this model, the flow resistance depends on the gap between RBC and vessel wall and on the plasma viscosity. Surface area (SA) and volume (V) of RBC from ten term neonates (N) and ten adults (A) were measured by means of a micropipette system and plasma viscosity (PV) was determined using a capillary viscometer. Neonatal RBC had larger V and SA than adult RBC (107 \pm 6 vs 90 \pm 4 fl, and 154 \pm 7 vs 137 \pm 7 μ m³). PV was lower in neonates (1.04 \pm 0.10 cP) than in adults (1.26 \pm 0.13 cP). The critical vessel diameter below that the gap becomes too small to allow sufficient lubrication is higher for neonatal RBC (3.3 μ m) than that for adult RBC (2.9 μ m). The driving pressure, hematocrit and viscosity of neonatal RBC is higher than that of adult RBC if both cell types have been suspended in the same medium. However, the driving pressure and viscosity of neonatal and adult RBC is similar for neonatal and adult RBC if the cells have been suspended in the corresponding neonatal and adult plasma. We conclude that the flow properties of neonatal and adult RBC in vessels with diameters of 3.5 to 6 μ m are not different since the larger size of neonatal RBC is compensated by a lower PV.

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EFFECT OF BLOOD TRANSFUSION ON BLOOD VOLUME, BLOOD VISCOSITY AND PERIPHERAL CIRCULATION IN NEONATES
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Peripheral blood flow (PBF) depends on blood pressure (P), blood viscosity (η) and vascular hindrance (Z):
 $PBF = P/R = P/(Z \cdot \eta)$, where R is the peripheral resistance. Thus, blood transfusion can increase PBF by increasing P and decreasing Z and decrease PBF by increasing η . We have studied PBF (venous occlusion plethysmography), P (Doppler), blood volume (BV, Evans blue) and η (capillary viscometer) in 14 anemic neonates (gestational age 27-42 weeks) during the first week of life immediately before and after transfusion (Tx) of 20 ml of whole blood.

($\bar{x} \pm SD$; * P<0.05)	pre Tx	post Tx
BV (ml/kg)	87.6 \pm 15.4	96.8 \pm 12.7*
Hct (l/l)	0.37 \pm 0.04	0.47 \pm 0.04*
η (cP)	2.9 \pm 0.3	3.6 \pm 0.3 *
PBF (ml/min \cdot 100ml)	8.9 \pm 2.4	11.9 \pm 2.5 *
P (mmHg)	73.0 \pm 16.1	82.6 \pm 19.1*
R (P/PBF)	8.6 \pm 2.0	7.6 \pm 1.6 *
Z (R/ η)	2.9 \pm 0.7	2.1 \pm 0.5 *

We conclude that Tx results in vasodilatation (decrease in Z) and increased pressure, thereby increasing blood flow in spite of increasing blood viscosity.

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CHANGES IN CEREBRAL PERFUSION DURING NEONATAL EXTRACORPOREAL MEMBRANE OXYGENATION. F. van Bel, F.J. Walther, E.S. Gangitano, J.R. Snyder. Univ. of Southern California, Huntington Memorial Hospital, Dept. of Neonatology, Los Angeles, U.S.A.

To further understand the pattern of cerebral blood flow during neonatal ECMO, we performed serial measurements of the peak systolic (PSFV), end-diastolic (EDFV), and mean blood flow velocities (MFV) of the anterior cerebral arteries in 8 infants pre-, during, and post-ECMO. Mean \pm SD values of the MFV increased from 9.4 \pm 2.4 cm/sec pre-ECMO to 13.0 \pm 2.2 cm/sec (p<0.01) during maximal bypass flow (91 \pm 17 ml/min/kg). Simultaneously, the mean EDFV increased from 4.0 \pm 1.9 to 7.2 \pm 1.7 cm/sec (p<0.01), the mean PSFV increased from 10.3 \pm 1.9 to 12.8 \pm 3.1 cm/sec (NS), and the pulsatility index (PI: PSFV-EDFV/PSFV) decreased from 0.63 \pm 0.12 to 0.45 \pm 0.05 (p<0.01). Post-ECMO values of MFV (8.4 \pm 2.5 cm/sec), EDFV (3.8 \pm 1.4 cm/sec), PSFV (9.3 \pm 2.5 cm/sec), and PI (0.60 \pm 0.08) were comparable to pre-ECMO values. During weaning from ECMO the PI increased steadily with a concomitant decrease of the MFV.

These data indicate that during neonatal ECMO cerebral perfusion is higher and cerebral vascular resistance lower than pre- and post-ECMO and that these changes are related to the amount of bypass used.

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Ophthalmic Blood Flow Velocity (OBFV) in Sick Neonates. Lindner W, Schindler M, Riegel K, Versmold HT*, Department of Pediatr. and Gyn. Ob.*, Univ. of Munich, F.R.G.

Abnormal pattern of cerebral BFV (CBFV) is known in neonates with diseases, associated with an increased risk of retinopathy of prematurity (ROP). No information is available on OBFV (cm/s) in sick neonates. We studied OBFV and CBFV by pulsed Doppler sonography in 21 preterm (median gest. age 28 wks, range 24-32) and 5 term neonates, with different problems, causing abnormal CBFV. (Patent ductus art.(PDA, n=16), Indomethacin(n=1), polycythemia(n=5), hypocalcemia (n=6), hydrocephalus(n=1). 30 normal neonates (1), matched for GA, served as controls. **Results:** Changes in OBFV were always related to changes in CBFV. No infant with abnormal CBFV had normal OBFV. PDA caused increased systolic and decreased or reverse diastolic OBFV. All other conditions were associated with a decrease in syst. and diast. OBFV. Mean OBFV decreased between 30 and 81 % in polycythemic infants. The decrease was usually more marked in OBFV than in CBFV, however in hypocalcemic infants mean OBFV was less decreased (2% / torr pCO₂) than CBFV (4% / torr pCO₂). **Conclusion:** Low OBFV is present in neonates with clinical conditions causing decreased CBFV, this may cause abnormal retinal perfusion. Thus, abnormal OBFV may be a risk factor for ROP. CO₂ reactivity of ophthalmic and cerebral arteries may be different. (1) Controls are partly presented in Pediatr Res 22:241A, 1987.

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RENAL HANDLING OF SIALIC ACID (SA)

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Sialic acid (SA) is mainly bound to glycoconjugates and is a component of glomerular membranes. Free SA is the most abundant negatively charged carbohydrate in urine. Salla Disease (SD) is a lysosomal storage disorder due to impaired egress of free SA from lysosomes. SD patients have 5-10 times elevated free SA in their urine and plasma.

We determined the renal clearance of SA in controls and SD patients over a broad range of filtered loads, using an HPLC method. All patients had a normal creatinine clearance or glomerular filtration rate (GFR). The SA clearance approximated the GFR in all subjects, regardless of filtered load; hence, the fractional excretion of SA was approximately 100%. These results suggest that free SA is filtered by the kidneys and not reabsorbed, which contrasts markedly with the renal handling of glucose, which is efficiently reabsorbed at plasma concentrations 1000 times that of free SA.

	Plasma SA (nmol/ml)	Filtered Load (nmol/min)	Fractional Excretion (%)
	($\bar{X} \pm SD$)	(range)	($\bar{X} \pm SD$)
Controls (n=9)	0.80 \pm 0.28	39-72	95.2 \pm 14.4
SD (n=8)	4.13 \pm 2.48	160-380	114.6 \pm 8.9