- 5 Micro- and macrocirculation in neonatal polycythemia
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Polycythemia is associated with an increased incidence of cerebral hemorrhage and neurodevelopmental abnormalities.

Material and Methods: Whole blood and plasma viscosity (capillary viscosimeter), RBC deformability (rheoscope) and aggregation (Myrenne Aggregometer), cerebral and peripheral blood flow (plethysmography and Doppler sonography) and plasma volume (Evans blue) were studied in 21 neonates with gestational ages of 35-41 weeks.

Results: The neonates were divided into two groups according to the hematocrit (group I: < 0.65 l/l, n=13; group II: ≥ 0.65 l/l, n=8)

Hematocrit (l/l)	0.31-0.64	0.65-0.75
Blood viscosity (V, cP)	3.1 ± 0.6 **	5.6 ± 0.9
Cerebral blood flow (CBF, ml/min/100g)	43 ± 1 **	30 ± 5
Vascular hindrance (P/CBF/V)	40.5 ± 7.4 *	34 ± 6.5
Cerebral RBC flow (CBF x Hct)	20.3 ± 1.8	20.5 ± 2.8
Peripheral blood flow (PBF, ml/min/100g)	8.0 ± 1.7 **	6.5 ± 1.2
Peripheral RBC flow (PBF x Hct)	3.9 ± 1.0 *	4.9 ± 0.8

P = art. pressure (mmHg); RBC = red blood cells; * p < 0.05, ** p < 0.005
Conclusions: Cerebral RBC flow is not affected by hematocrit, plasma volume or blood volume. The effect of V on vascular resistance is partially counterbalanced by decreasing vascular hindrance (i.e. by vasodilatation).

- 6 PROGNOSTIC FACTORS IN THE HEMOLYTIC-UREMIC SYNDROME.
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During 1972-1986 142 children were treated. Most of them were infants (73%). Prodromal symptoms lasted 1-16 days and usually involved the gastrointestinal tract. Severe gastrointestinal symptoms had poor prognostic significance. Convulsions, coma and hypertension which did not resolve after correction of hypervolemia were also features of poor prognosis. Laboratory findings at onset such as hyperkalemia exceeding 7 mmol/L and acidosis with bicarbonate levels below 12 mmol/L were associated with a worse prognosis. 80% of children required peritoneal dialysis. Delay in the starting of dialysis after 5th day of anuria was a further poor prognostic factor. The total mortality rate reached 25.4%. This high mortality rate was due to the severe clinical course, to the late diagnosis and delay in dialysis treatment. The greatest risk of death existed in the first 3 weeks from onset. They were followed up from several months to 14 years. Twelve children developed chronic renal failure (11.3%). Discriminant analysis revealed the following factors to be associated with the development of chronic renal failure: delay in dialysis, onset of disease in spring/summer months, older age, duration of acute renal failure, older age, duration of hemolysis and peak level of creatinine.

- 7 URINARY EXCRETION OF BETA-2-MICROGLOBULIN IN CHILDREN WITH URINARY TRACT INFECTIONS
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The aim of this study was to evaluate, if urinary Beta-2-Microglobulin (B2M) excretion is useful in the level diagnosis of urinary tract infections (UTI).

161 children with significant bacteriuria were studied. B2M was measured in 24-hrs urine collection (ph>6) by solid-phase-radioimmunoassay (B2M-monoclonal RIA KIT Prague). The samples were studied at the onset of the disease and after antibacterial treatment. The excretion of B2M was significantly higher in patients with upper UTI than in those with lower UTI or asymptomatic bacteriuria. Especially high values were observed among children with upper UTI and with accompanying radiological changes. After antibacterial treatment the excretion of B2M decreased significantly in all patients with UTI. The sensitivity of urinary B2M as a predictor of parenchymal involvement was 89.4% with a specificity of 96.5%.

Conclusions: 1) B2M excretion helps to distinguish between upper and lower UTI in children. 2) High values of B2M excretion in children with UTI are highly suggestive of parenchymal involvement, particularly in cases with radiological changes.

- 8 SERUM MICROPROTEINES [α_1 AND β_2 -MICROGLOBULIN (MG)] FOR ESTIMATION OF FETAL GLOMERULAR RENAL FUNCTION
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Serum microprotein levels can be used to predict glomerular renal function. To evaluate microprotein determination to estimate fetal renal function, levels of α_1 - and β_2 -MG and creatinine were determined in cord blood of 183 healthy term newborns (A), in both cord blood (CB) and maternal blood (MB) of 66 preterm neonates, GA 28-36 weeks, (B) and in cases with impaired maternal or fetal renal function.

Results: A: values for healthy term neonates (n=183):

	β_2 MG (mg/l)	α_1 MG (mg/l)	Creatinine (mg%)
x	2.57	22.4	0.71
SD	0.56	4.16	0.12

B: preterm neonates (n=66):

B. Preterm neonates (n=66)						
	β_2 MG (mg/l)		α_1 MG (mg/l)		Creatinine (mg%)	
	CB	MB	CB	MB	CB	MB
x	3.37	1.81	27.2	38.1	0.60	0.54
SD	0.64	1.59	6.2	13.0	0.24	0.25

Microprotein levels depend on gestational age and are higher in preterm than in term infants. They are independent of maternal renal function which is distinct in cases with severe maternal or fetal renal insufficiency, for example in Potter's syndrome: (n=4) CB: Crea .7-.9 mg%, β_2 -MG 14.9-16.6; α_2 -MG 160-180 mg/l. **Conclusion:** Microprotein determination in fetal blood can be used to detect severe renal function disturbances and to estimate glomerular filtration rate independently of maternal renal function.

- 9 RENAL VERSUS CEREBRAL BLOOD FLOW VELOCITIES IN PRETERM INFANTS WITH PATENT DUCTUS ARTERIOSUS
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Angiographic and animal studies have shown that most of the diastolic flow in the PDA originates from the descending aorta, potentially leading to abdominal ischemia. We studied blood flow velocities directly in renal (RA) and internal carotid arteries (CAI) of 10 infants (body weight 620-1485g, gestat. age 26-33 wks) with PDA and after surgical ligation by Doppler ultrasonography. Peak systolic (Vs), enddiastolic (Vd), time averaged maximal velocities (TAM), resistance indices (RI) were determined.

Results (cm/sec):		Vs	Vd	RI	TAM
PDA group	RA	90 ± 25	-11 ± 9	1.11 ± 0.06	14 ± 5
post PDA	RA	46 ± 9 *	6 ± 3 *	0.87 ± 0.07 *	19 ± 5 *
PDA group	CAI	36 ± 10	-1 ± 4	1.02 ± 0.10	12 ± 5
post PDA	CAI	29 ± 8 *	5 ± 3 *	0.85 ± 0.07 *	13 ± 5

* p < 0.05 (Wilcoxon test)

With PDA, Vd was markedly decreased in the RAs and even reversed in 7/10 infants. In the CAI, however, Vd was retrograde only in 3/10 infants and TAM did not show any difference with surgical ligation. Our findings indicate that a PDA may also result in a steal of blood away from the kidneys. This is more pronounced than in the CAI.

- 10 INVESTIGATION OF THE UROEPITHELIAL DEFENSE MECHANISM IN HEALTHY FEMALE AND CHILDREN WITH RECURRENT URINARY TRACT INFECTIONS (UTI)
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A MOST DEFENSE DEFECT ON THE LEVEL OF THE UROEPITHELIAL CELL (UEC) HAS BEEN DESCRIBED IN PATIENTS WITH RECURRENT UTI, WHEREAS

HEALTHY PERSONS REVEAL A POTENT ANTIBACTERIAL CAPACITY OF THEIR UEC IN VITRO. THE ANTIBACTERIAL EFFECT IS DEPENDENT ON TIGHT CONTACT BETWEEN BACTERIA AND UEC SURFACES. WE INVESTIGATED RECEPTOR-LIGAND MEDIATED TRANSMEMBRANOUS SIGNALS IN E. COLI-UEC COCULTURES. 10⁵ UEC AND 10⁶ E. COLI (K1, O1, H7, PILI II POS. STRAIN)/ML RPMI MEDIUM WERE COCULTURED FOR 15, 30, 45 MINUTES AT 37°C. ALIQUOTS WERE DILUTED AND SUSPENDED INTO AGAR PLATES; BACTERIAL COLONIES WERE ALLOWED TO GROW FOR 24H. THE INFLUENCE OF DIFFERENT PHARMACOLOGICAL SUBSTANCES ON THE E. COLI-UEC INTERACTION WAS INVESTIGATED. OUR RESULTS DEMONSTRATE THAT INHIBITION OF BACTERIAL ADHESION TO UEC BY SP 54 SIGNIFICANTLY DIMINISHED THE SUPPRESSIVE CAPACITY OF HEALTHY UEC. SIMILAR RESULTS WERE OBSERVED WHEN CALCIUM ANTAGONISTS (VERAPAMIL) OR CALMODULIN BLOCKING SUBSTANCES (OXATONIO) WERE ADDED TO THE E. COLI-UEC COCULTURES. HOWEVER, ACTIVATION OF SECOND MESSENGER MOLECULES BY EPINEPHRIN OR THEOPHYLLIN AND FORSCOLIN (BOTH KNOWN TO INCREASE INTRACELLULAR C-AMP LEVELS) COULD RESTORE THE FORMERLY DEFICIENT ANTIBACTERIAL DEFENSE FUNCTION IN UTI-UEC. PROSTAGLANDINS AS MEDIATORS IN INFLAMMATORY REACTIONS (PGI₁, PGF_{2α}) ALSO ACTIVATED UTI-DEFICIENT UEC TO SUPPRESS E. COLI GROWTH. IN CONTRAST, THE ADDITION OF INDOMETHACIN ABOLISHED THE ANTIBACTERIAL EFFECT OF HEALTHY UEC. WE CONCLUDE THAT TIGHT CONTACT BETWEEN E. COLI AND UEC DELIVERS METABOLITES OF PROSTAGLANDINS FROM THE CELL MEMBRANE WHICH WILL INDUCE SECOND MESSENGER ACTIVATION RESULTING IN A SIGNIFICANT BACTERIAL GROWTH SUPPRESSION.