

29

URINARY MEASUREMENTS OF ATRIAL NATRIURETIC PEPTIDE (ANP), CYCLIC GUANOSINE MONOPHOSPHATE (cGMP), ARGININE-VASOPRESSIN (AVP) AND ALDOSTERONE (ALDO) IN NEWBORN INFANTS.

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Elevated ANP plasma levels may cause the physiological weight loss during the first days of life. We investigated the urinary ANP, cGMP, AVP and Aldo excretion in term newborn infants for the first 5 days of life. We measured plasma ANP, cGMP and AVP levels in cord blood of 13 healthy neonates. Urine was collected from these infants and 8 other neonates during the first 5 days of life to determine the excretions of ANP, cGMP, AVP, Aldo and sodium. Also older infants were studied (aged 45-387 days). Plasma levels of ANP (median  $n=173$  pg/ml), cGMP ( $n=4.8$  pmol/ml), and AVP ( $n=266$  pg/ml) were significantly higher in cord blood than in venous blood of older children. cGMP excretion was significantly higher on day 1 ( $p<0.001$ ), day 2 ( $p<0.05$ ) and day 3 ( $p<0.05$ ) than in control infants. Na excretion (day 1+2: $p<0.001$ ; day 3: $p<0.05$ ) and urine volume (day 1-3: $p<0.05$ ) were significantly lower during the first 3 days of life than in controls. ANP, Aldo and AVP excretions did not show an age dependence. There were no relations between sodium excretion and ANP, Aldo or AVP. Only cGMP excretion correlated with volume excretion on day 1 ( $r=0.77$ ;  $p<0.001$ ), day 2 ( $r=0.88$ ;  $p<0.001$ ) and day 5 ( $r=0.59$ ;  $p<0.05$ ). There was a significant correlation between cord plasma cGMP levels and (1) sodium excretion on day 1 ( $r=0.67$ ;  $p<0.02$ ), and (2) weightloss from day 1 to day 2 ( $r=-0.54$ ;  $p<0.05$ ). Conclusions: 1. In neonates only urinary cGMP levels show an age dependence among the investigated hormones. 2. Excretion of cGMP, but not of ANP, Aldo or AVP, may reflect volume homeostasis during the first 5 days of life. 3. cGMP plasma levels seem to relate with sodium excretion and weight loss in early infancy.

30

PLASMA ATRIAL NATRIURETIC PEPTIDE (ANP) AND CYCLIC GUANOSINE MONOPHOSPHATE (cGMP) LEVELS AND ANP-BINDING SITES ON PLATELETS IN THE PERINATAL PERIOD. J. Weil, T. Strom, A. Timnik, R. Gerzer\*, Dept. of Pediatrics and Medicine\*, University of Munich (FRG).

We reported higher plasma ANP-levels in newborns than in older children. It was speculated that elevated ANP-levels may result in a down-regulation of ANP-binding sites. To prove this hypothesis we measured ANP together with the second messenger cGMP and ANP-binding sites on platelets in the perinatal period. Plasma ANP- and cGMP-levels were determined in mothers after vaginal delivery ( $n=21$ ) and after sectio ( $n=7$ ) and in umbilical venous ( $n=27$ ) and arterial ( $n=15$ ) cord blood of term newborns. ANP-binding sites on platelets were determined in cord blood. ANP- and cGMP-levels were significantly higher in mothers and in cord blood than in non-pregnant women ( $n=11$ ) and in control infants beyond the neonatal period, resp. ( $n=18$ ,  $p<0.01$ ). There was a linear correlation between ANP- and cGMP-levels ( $r=0.7$ ). There were no significant differences in ANP-binding sites/platelet and Kd between newborns and healthy adults ( $n=19$ ). Our results do not support the hypothesis of a down-regulation of "ANP-receptors" in the perinatal period. The elevated cGMP levels in patients with high ANP-levels agree with the concept that cGMP is a Marker for the cellular effects of ANP.

31

Atrial Natriuretic Peptide (ANP) concentrations in infants and children receiving intensive care.

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ANP concentrations were measured in 38 children undergoing intensive care. 25 were neonates; 24 with respiratory distress syndrome (RDS) and 5 with patent ductus arteriosus (PDA). 6 infants with arrhythmias and 7 children who underwent the Fontan procedure were also studied. In all groups median ANP concentration was high, although values fluctuated in patients with RDS or PDA (median 360 pg/ml, range 0 - 2666 pg/ml, normal range 90 - 240 pg/ml). In these 2 groups there was no significant correlation with plasma or urine sodium concentrations, or fluid intake. There was a significant positive correlation with arginine vasopressin (AVP) concentrations ( $p<0.001$ ). Treatment of the arrhythmias produced a fall in ANP concentrations (median before and after: 500 pg/ml and 165 pg/ml,  $p<0.01$ ). The Fontan procedure produced a significant rise in ANP concentrations (median before and after: 149 pg/ml and 283 pg/ml,  $p<0.005$ ). We conclude that sick infants and children often have strikingly elevated ANP concentrations, especially in conditions associated with an increase in right atrial dimensions, but the diagnostic value of ANP measurement remains to be evaluated. The positive correlation with AVP concentrations was unexpected, and is being further investigated.

32

THE INFLUENCE OF ARTIFICIAL COLONIZATION WITH THE NON-ENTEROPATHOGENIC E. COLI STRAIN O83 ON THE INTESTINAL FLORA IN INFANTS FROM BIRTH UP TO 6 MONTHS. MILADA SLAVIKOVÁ, RAJA LODINOVÁ-ŽADNÍKOVÁ, LARS Å. HANSON.

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In four groups of infants 1) breast-fed colonized, 2) formula-fed colonized, 3) breast-fed controls, 4) formula-fed controls the dominant bacterial strains were isolated and the presence of adhesins followed by agglutination. The selected strains were serotyped and the adherence tested on the cell line HT-29. Breast-fed infants had less P fimbriated and more haemagglutination negative isolates than the bottle-fed infants. Also the total HT-adherence of P fimbriated isolates and t1 isolates was lower in this group of infants. The E.coli strain O83 is t1 fimbriated - the total adherence of E.coli O83 isolates was higher in breast-fed colonized, than in bottle-fed colonized infants and also the % of E.coli O83 isolates was higher in breast-fed infants. In the colonized groups the % of the other t1 fimbriated isolates was lower than in the control groups.

33

SHIGA-LIKE TOXIN PRODUCING ESCHERICHIA COLI ASSOCIATED WITH ULCERATIVE COLITIS.

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Ulcerative colitis (UC) resembles clinically and histologically the newly recognized hemorrhagic colitis which has been etiologically linked to colonic colonization with Shiga-like toxin (SLT) producing E.coli O157:H7. This prompted us to investigate a possible common infectious origin.

SLT-producing non-O157 E.coli were identified in the feces from 4 out of 10 patients aged 10 to 35 years with exacerbating UC but not in juvenile patients with active Crohn's disease ( $n=5$ ) using colony blot hybridization tests with SLT-I and SLT-II gene specific oligo-DNA probes. Stool filtrates in 5 of 7 cases of UC were cytotoxic for Vero and HeLa cells (titers 1:20 to 1:512). Two patients were treated antibioticly, and clinical remission with concomittant elimination of SLT producing strains was observed.

Our findings suggest that SLT producing bacteria may play a role in supporting or exacerbating UC and that antiinfectives may be of therapeutic value in some patients.

34

IS CANDIDA ACQUISITION IN THE INTENSIVE CARE NEONATE DURING ANTIBIOTIC TREATMENT RELATED TO GESTATIONAL AGE? A PROSPECTIVE STUDY.

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In neonates antimicrobial treatment is an important factor to acquire Candida, with risk of subsequent systemic infection. We prospectively studied the acquisition of candida, in term vs. preterm infants, who were treated with ampicillin in combination with an aminoglycoside.

185 infants were studied; infants who acquired candida before antibiotic treatment, were excluded ( $n=13$ ). The remaining infants were grouped according to their gestational age.

Of the 172 infants, 31 acquired candida (=18%) 3 of the latter developed systemic candidiasis.

Gestational age (wk)	Total	No. with candida(%)	candidiasis
< 32	76	16 (21)	3
≥ 32 < 36	55	9 (16)	
> 36	41	6 (11)	

Conclusion: There is no significant difference in incidence in Candida acquisition between the different gestational age groups. In particular infants with a gestational age of 32 weeks or less seem to be at risk for systemic candidiasis.