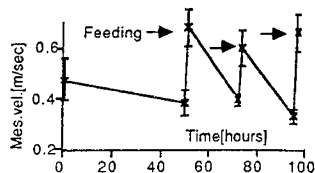


**Feeding increases mesenteric blood flow velocity in healthy term newborn infants.**  
Martinussen M., Linker D., Vik T. and Brubakk A.M., University Hospital, Trondheim, Norway.

The aim of the study was to evaluate the effect of feeding on mesenteric blood flow velocity during the first week of life.

Blood flow velocity was recorded using a duplex ultrasound scanner and pulsed Doppler, with angle correction when necessary. Measurements were made in the superior mesenteric artery immediately after its departure from the aorta in eleven term infants within the first hour after birth, and before and after feeding on the third, fourth and fifth days.

Baseline mesenteric blood flow velocity did not change during the first five days of life. However, there was a significant increase in blood flow velocity after feeding (fig).



Our findings suggest that normal newborns are able to increase the mesenteric blood flow in response to feeding even during the first days of life. This must be taken into account when studying mesenteric blood flow during disease in the newborn period.

**Prolonged preoperative ventilation prevents persistent fetal circulation (PFC) in congenital diaphragmatic hernia (CDH).**

Brubakk AM, Linker D, Eik-Nes S, Kulaas T, Vik T, Haugen SE, University Hospital, Trondheim, Norway.

CDH has a very high mortality rate (60 - 80 %), with the main cause of death being PFC in the postoperative stage. Intrauterine recognition of the disease with vigorous resuscitation after birth has not improved the prognosis.

We decided to ventilate the infants with CDH, monitor the pulmonary artery pressure (PAP) and delay operation until the PAP had approached normal. Ten infants with CDH were admission to our unit. Two infants died shortly after admittance. In the remaining eight PAP was estimated by doppler ultrasound to 50 - 90 mm Hg on admission and decreased slowly to near normal range (30 - 50 mm Hg) in 3 to 21 days. Following operation none of the eight infants developed PFC and all survived.

We conclude that prolonged preoperative ventilation until pulmonary hypertension has resolved prevents PFC and improves the prognosis of CHD.

**X-linked centronuclear myopathy: gene localization and prenatal diagnosis**

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Departments of Pediatrics, University of Berne (1) and Zürich (3), and Department of Human Genetics, University of Würzburg (2).

The X-linked recessive centronuclear myopathy (XLR-CNM), a severe neonatal disorder characterized by generalised hypotonia, muscle weakness and primary asphyxia, has recently been mapped to Xq28. We investigated 70 members of eight CNM-families using the Xq28 probes DX13, St14, FVIII, 767, and cpX67. Strong linkage was found with St14 (lod score  $z = 3.12$ ;  $\Theta = 0.01$ ), followed by FVIII, DX13, and 767, proving them to be useful for prenatal diagnosis (pnD). This is the first report on pnD in two CNM-families: In family 1 two pnD have been performed for an obligate carrier having had two affected sons. Linkage analyses using probes DX13, St14, and 767 showed a male fetus having inherited the same X-chromosome as his two deceased brothers, and the parents choose abortion. In a further pregnancy the fetus was a girl having no risk of being affected. The patient of family 2 was a sporadic case and the mother's carrier state was not known. pnD in the second pregnancy detected that the fetus had inherited the grandpaternal X-chromosome as his deceased brother. At birth the boy was affected, proving that pnD was correct and that the CNM-mutation had occurred during the grandpaternal spermiogenesis. This presentation points to the fact, that in families at risk for CNM pnD can now be offered.

**SEPARATION OF URODILATIN AND ANP IN THE URINE OF HEALTHY NEWBORNS, INFANTS, AND YOUNG ADULTS.** Bauer K, Solc J\*, Timik A, Solcova A\*, Weil J. Depts. of Pediatrics, Univ. of Munich, FRG and Univ. of Plzen\*, CSR

ANP immunoreactive protein (RIAtotal) in urine does not represent alpha-h-ANP CDD ANP-99-126 (ANP), but contains at least one other ANP-like peptide, called urodilatin CDD ANP-95-126 (URO). The percentages of ANP and URO in urines of different populations are not known. We separated URO and ANP by HPLC and then determined the concentrations of the 2 fractions by ANP-RIA. We report first results of this technique in 6 healthy neonates (age 2-19 d), 4 infants (age 1-10 mo) and 10 male volunteers (age 19-22 y).

Results (+SD)	adults	neonates	infants
Diuresis (ml/kg d)	17+9	80+23**	59+28
RIAtotal (pg/ml)	40+21	18+4*	13+4*
RIAtotal (pg/kg d)	777+840	1526+668	812+338
ANP (% of total)	31+6	29+3	33+9
URO (% of total)	24+5	25+3	25+5

\*\*: $p < 0.001$  \*: $p < 0.01$  vs adults

RIAtotal concentration was higher ( $p < 0.01$ ) in adults than in newborns and infants, but RIAtotal excretion (pg/kg d) did not differ between groups. ANP and URO (% of total) did not differ between groups, about 45% of RIA total were not found in the ANP- and URO-fractions. There were no correlations between concentrations of RIAtotal, ANP, URO and diuresis, sodium excretion or  $FE_{Na}$ .

Conclusion: The percentages of URO and ANP did not differ between newborns, infants and adults. The role of urinary ANP and URO and the nature of the proportion of RIAtotal not found in the ANP- and URO-fractions need further investigation.

**RECOMBINANT HUMAN ERYTHROPOIETIN (rhEPO) STIMULATES TESTOSTERONE PRODUCTION ON ISOLATED ADULT RAT LEYDIG CELLS.**

R. Microni, \*G. Montini, \*G. Zaccarello, C. Foresta.

Sponsored by: Sergio O. Saia

III<sup>rd</sup> Chair of Medical Pathology - \* Pediatric Department - Padua - I

Erythropoietin (EPO), a glycoprotein considered a growth factor, corrects anemia of renal patients. These patients improve their sexual functions after EPO treatment. Recent data demonstrate that several growth factors act, through a specific receptors, on Leydig cells influencing steroidogenesis. The aim of our study is to evaluate the effect of rhEPO on testosterone secretion by isolated adult rat Leydig cells. Aliquots of high purified rat Leydig cells (90%-92%), by Percoll discontinuous gradients, were incubated in M-199 with L-glutamine, Hanks' salt, BSA 0.2%, at 34°C in controlled atmosphere, in shaking bath room, in sterile polyethylene tubes containing rhEPO (from 50 nU to 50 U/ml). After 3h the incubation was stopped and tubes were immediately centrifuged 1500g/15min and supernatant was stored at -20°C until testosterone assay by RIA method. Results: rhEPO exerts a significant ( $p < 0.05$ ) stimulatory effect on testosterone secretion, starting at the dose 1.00 U/ml, with the maximal effect at the dose of 10.0 U/ml ( $6.8 \pm 1.2ng/2 \times 10^6 cells/ml/3h$  versus control  $3.5 \pm 0.7$ ). Conclusions: Our preliminary data show, for the first time, that rhEPO influences rat Leydig steroidogenesis enhancing testosterone production. These results suggest that rhEPO therapy could improve sexual function involving also Leydig steroidogenesis.

**RESULTS OF ELECTROPHYSIOLOGICAL INVESTIGATIONS IN CHILDREN WITH HIV-INFECTIONS**  
Seeger, Jürgen, Jacobi, Gert, Schmitt, Bernhard\*, Dep. of Pediatric Neurology, J.W. Goethe- Univ., Frankfurt, FRG; \*EEG-Dep., Kinderspital, Zürich, CH.

Electrophysiological studies were done to investigate the value of EEG and evoked potentials (AEP, VEP, SEP) in detecting CNS affections in HIV-infected children staged P0-P2 (CDC-classification). Among 90 EEGs from 38 patients only 5 showed slowing of background activity (from 4 patients of the P2-group). In 38 AEPs of 27 patients staged P1 and P2 we found prolongation of interpeak-latencies only in the P2 group (7 of 20 patients). Late onset of AEP was found in the P1 (4/7) and the P2 (2/20) group. 37 VEPs in 26 P1/P2 patients were abnormal (peak latency and amplitude) in 2 of 7 (P1) and in 4 of 19 (P2) patients. SEP (25 from 22 P1/P2 patients) showed an amplitude reduction of the cortical potential (N20) only in 2 of 6 P1 patients, 16 P2 patients had normal results. Electrophysiological investigations can be helpful in detecting CNS affections before the appearance of clinical symptoms, but in the most cases the pathological findings are unspecific and neurological deterioration can occur despite normal findings.