● 25

Klaus Børch and Gorm Greisen. Dept. of neonatology, University Hospital, Copenhagen, Denmark.

WIDESPREAD REGIONAL CEREBRAL BLOOD FLOW DISTURBANCES IN PRETERM INFANTS WITH INTRACEREBRAL HEMORRHAGES

Intracerebral hemorrhage (ICH) possibly induces damage to surrounding cerebral tissue by ischaemia which may cause severe neurological sequelae, mainly cerebral paresis.

Methods: We have measured regional CBF within the first days after diagnosis (1-3days) in 6 preterm infants (GA 26-29weeks) with unilateral ICH and in 9 preterm infants (GA 26-32weeks) without ICH using single photon emission computer tomography and ^{99m}Tc-HMPAO (Ceretec®) i a dose of 4MBq/kg. We used a 4-head fast rotating gamma camera (Tomomatic 248®) with a spatial resolution of 6x6x8mm.

Results: Compared to the control group the flow to the white matter of the affected hemisphere was decreased 37% (p=0.0003, non-paired t-test). The flow to the other regions did not differ significantly but the flow to the grey matter of both hemispheres and to the basal ganglia of the affected hemisphere showed a higher variation than the control group (p<0.01, F-test).

Conclusion: Similar to former studies in premature infants we have demonstrated a decreased white matter flow in the affected hemisphere. In contrast, flow abnormalities was seen in the grey matter of both hemispheres. This indicates that ICH may be a part of a more widespred brain damage and may explain the variation in neurological outcome.

● 26

ADRENERGIC α-RECEPTORS IN THE HEART: DO THEY MATTER?

Kjell Borthne[1 & 2], Per Hågå[3], Asbjørn Langslet[3], Jan-B. Osnes[1], Tor Skomedal[1].
[1]Dept. of Pharmacology, University of Oslo. [2]Inst. for Surg. Research, Rikshospitalet, Oslo. [3]Children´s Dept., Ullevål Sykehus, Oslo.

The relationship between α- and β-adrenoceptors as mediators for the myocardial inotropic response was investigated ex vivo in right atrial preparations from the cannulation site for cardio-pulmonary bypass during surgery for congenital heart defects. Contractile force was measured in physiological organ baths during electrical stimulation.Such preparations may be used for several types of ex vivo studies on myocardial function.

We reversed a near maximal inotropic response to endogenous noradrenaline with β-adrenoceptor antagonist timolol and α_1-adrenoceptor antagonist prazosin. Median 77% (range 94% to 42%) was mediated by β-adrenoceptors and median 23% (range 6% to 58%) by α_1-adrenoceptors.

Stimulation of α- and β-adrenoceptors evokes different intracellular signalling mechanisms with different patterns of Ca^{++} handling. The resulting types of inotropic responses are different. α_1-adrenoceptor stimulation also stimulates intracellular alkalinisation and mechanisms as activated by ischemic preconditioning.

● 27

EFFECTS OF DEXAMETHASONE TREATMENT ON DIURESIS, SERUM UREA AND ARTERIAL BLOOD PRESSURE IN PRETERM INFANTS WITH BRONCHOPULMONARY DYSPLASIA.

Arend F. Bos, Willemina W.C. Reitsma-Bierens, Albert Okken. Dept. of Pediatrics, Beatrix Children's Hospital, Groningen.

The influence of dexamethasone on diuresis in preterm infants has not well been studied. In 15 preterm infants (gestational age 27.7±0.9 weeks, birthweight 994±192 grams, mean ± SD) with bronchopulmonary dysplasia we measured urine output, serum urea and arterial blood pressure (ABP) on the day before, and on 4 consecutive days after starting treatment with dexamethasone (0.6 mg/kg/d, twice daily). Diuresis did not change, until after 48 hours a substantial increase in diuresis occured. During the study period fluid and energy intake remained constant. Blood glucose levels did not change. Results are summarized in the table, data are expressed as mean ± SD.

	day before dexa	day1	day2	day3	day4
diuresis (ml/kg/d)	117±15	124±23	108±29	147±27**	145±29**
fluid intake (ml/kg/d)	159±17	160±20	162±16	164±26	165±20
serum urea (mmol/l)	1.1±0.8	1.3±1.0	2.1±1.4*	4.5±2.5**	5.7±2.6**
systolic ABP (mm Hg)	49±10	53±13	57±14*	62±14**	65±11**
diastolic ABP (mm Hg)	29±5	32±9	32±10	39±9**	41±7**

* p<0.05, ** p<0.01, day2 etc. vs. day before dexa.

CONCLUSION: These results indicate that the significant increase in diuresis after 48 hours of dexamethasone treatment might be caused by 2 factors: (1) an increase of the osmolar load to the kidney due to the increase of serum urea; (2) pressure diuresis induced by the increase in arterial blood pressure.

▲ 28

EFFECTS OF DEXAMETHASONE TREATMENT ON SPONTANEOUS MOTOR BEHAVIOUR IN PRETERM INFANTS WITH BRONCHOPULMONARY DYSPLASIA.

Arend F. Bos, Roelie M. v.Asperen, Heinz F.R. Prechtl, Albert Okken. Dept. of Pediatrics, Beatrix Children's Hospital, Groningen.

In animals administration of corticosteroids during fetal life gives rise to a reduction in brain growth and altered animal behavior. The effect of corticosteroids on the developing human brain is not clear. We determined the quantity and quality of spontaneous movements (general movements -GM's-, twitches -TW-) in 15 preterm infants (gestational age 27.7±0.9 weeks, birthweight 994±192 grams) which were treated with dexamethasone for 1 to 6 weeks starting at the 2nd-4th week because of a severe bronchopulmonary dysplasia. Video-recordings of one hour were made prior to the course of dexamethasone therapy, after 24 and 48 hours of treatment, and subsequently weekly. Results on quantity are summarized in the table.

	before dexa	day 1	day 2	week 1
GM (number/hour)	38	28**	32	27**
GM duration (sec)	15	9**	9*	12
TW (number/hour)	34	13**	10**	17**

Data shown as median, * p<0.05, ** p<0.01 vs. day before dexa.

The quality of general movements impaired dramatically after starting the course of dexamethasone therapy, on day 2 even more than on day 1. The GM's became slower and monotonous, with a reduction in complexity and elegance. This effect remained during and even after treatment.

CONCLUSION: Our data demonstrate that dexamethasone treatment in preterm infants indeed effects brain function. The meaning of these findings for the further psychomotor development is not yet clear.

● 29

PLASMA AMINO ACID LEVELS IN PRETERM INFANTS WITH BRONCHOPULMONARY DYSPLASIA ON THE FIRST DAYS OF DEXAMETHASONE TREATMENT.

Arend F. Bos, Roelie M. v. Asperen, Dirk-Jan Reijngoud, Albert Okken. Dept. of Pediatrics, Beatrix Children's Hospital, Groningen.

Plasma levels of several amino acids are increased in preterm infants after several days of treatment with dexamethasone. Acute effects, however, have not been published. We measured plasma amino acid levels immediately before (day 0), 24 hours after and 48 hours after starting treatment with dexamethasone (0.6 mg/kg/day, twice daily) in 8 preterm infants (gestational age 26-29 weeks, birthweight 790-1485 grams) with bronchopulmonary dysplasia. Protein intake ranged from 1.1 to 2.9 g/kg/d. At 24 hours a substantial rise of nearly all amino acids was found. There was no relationship of protein intake on consecutive days or between individuals and rise in amino acid levels. Results are summarized in the table. ALA=alanine, GLU=glutamate, GLN=glutamine, LEU=leucine, ILE=isoleucine, VAL=valine, EAA=essential amino acids, TAA=total aminoacids. Data are shown as median, (range); fractional rise at 24 and 48 hours are given.

	day 0 (umol/l)	rise at 24 hrs	rise at 48 hrs
ALA	149 (55-502)	2.18*	2.33*
GLU+GLN	335 (89-660)	2.42*	2.41*
LEU+ILE+VAL	195 (99-360)	1.40*	1.41
EAA	660 (458-1530)	1.29*	1.47*
TAA	2105 (1122-5126)	1.59*	1.67*

* p<0.05 24 and 48 hours vs. day 0.

CONCLUSION: These results indicate that immediately following dexamethasone treatment protein catabolism occurs, presumably by an increased breakdown of muscle protein.

▲ 30

GROWTH FAILURE AND BODY COMPOSITION IN CHILDREN WITH INFLAMMATORY BOWEL DISEASE

Jan Bouquet, Anne M. Boot, L.K.Ragu Shanbhogue, Gineke Liefaard.
Sophia Childrens Hospital, Rotterdam, The Netherlands.

Malnutrition and growth failure are complications of inflammatory bowel disease (IBD) in children. Contributing factors to these effects are malabsorption, inadequate food intake, enhanced loss of nutrients, and corticosteroid treatment. Increased resting energy expenditure (REE) may also contribute to the growth retardation. Twenty children with endoscopicaly and histologically proven ulcerative colitis (n=13) or Crohns disease (n=7) and twenty age and sex matched controls were studied. REE was measured using a metabolic cart with principle of indirect calorimetry (Deltatrac, Datex, Finland), lean body mass (LBM) with bioelectrical impedance analysis (BIA). With Dual energy X-ray absorptiometry (DEXA) (Lunar Rad. Corp.,USA) an analysis of bone mineral density (BMD) and of LBM was done. The median age of IBD children was 13,5 years (range 6-18), of controls 14 years (range 6-18). The mean REE in children was increased: 33±1,7 versus 28,9±1,5 Kcal/kg bodyweight (p<0.01). The LBM was significantly increased in children with IBD versus controls: 81,5±1,7 versus 74,7±1,6% of bodyweight. REE in Kcal/kg LBM was not different in children with IBD versus controls: 41,7±2,1 versus 38,8±2,1 Kcal/kg. No difference in REE was found between patients with low (n=14) and high disease activity (n=6). In five patients (25%) a decreased BMD (>2 SD below mean value) of lumbar spine was found. An inverse relation between BMD and cumulative prednison dose was found. The LBM values measured with BIA and with DEXA technique correlated very well (R=0.95).