● 223

URINARY ENDOTHELIN-1 EXCRETION IN FUROSEMIDE-TREATED HUMAN NEONATES

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The present study was carried out to determine the response of urinary ET-1 excretion to furosemide administration in human neonates. Ten newborn infants with mean birthweight of 2752 g and mean gestational age of 37.1 weeks were given furosemide in a dose of 1 mg/kg. Prior to and following furosemide therapy urine was collected for a period of 12 hr and analyzed for creatinine, osmolality, sodium and potassium as well as for AVP, aldosterone and ET-1. Inresponse to furosemide administration urine flow rate and urinary osmolar, sodium and potassium excretion increased significantly, whereas creatinine excretion remained unchanged. Furthemore, following furosemide therapy there was an increase in AVP (19 ± 5.4 vs 28.7 ± 7.8 pg/kg/hr, p=0.06, NS), aldosterone (507 ± 120 vs 751 ± 203 ng/kg/hr, p<0.05) and ET-1 ($36.0\pm5,6$ vs 61.4 ± 8.7 fmol/kg/hr, p<0.05) excretion, respectively. Urinary ET-1 excretion was found to correlate positively with diuresis (r=0.75, p<0.001), sodium (r=0,53, p<0,0025), osmolar (r=0,73, p<0.001)and AVP excretion (r=0,72, p<0.001)but not with aldosterone excretion (r=0.10, p=0.96) It is concluded that the furosemide-induced diuresis and natriuresis is associated with significantly increased urinary ET-1 excretion which may further inhibit sodium and water reabsorption in the distal nephron.The enhanced generation of ET-1 in the renal medulla appears to be related to decreased medullary tonicity and AVP stimulation.

● 224

PURINES IN HYPOXEMIA/ISCHEMIA-REOXYGENATION IN PIGLETS.

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In order to study hypoxemia/ischemia-reoxygenation we devised 2 distinct models in 9-14 day old piglets: In model A, group (gr) 1 was subjected to 3 x 10 min severe hypoxemia by breathing 9% 0_2 alternating with 3 x 10 min reoxygenation with room air. In gr 2 cerebral ischemia was superimposed by ligating both carotides. In model B piglets were exposed to mild hypoxemia by breathing 11% 0_2 either continuously in 3 hours (gr 1) or repetitively in 3 x 1 hour, interrupted by 20 min room air breathing (gr 2). Hypoxanthine (Hx), xanthine and uric acid were measured in vitreous humor (VH), plasma and urine. Blood pressure (BP), heart rate and blood gases were monitored.

Hx(µmol/l):	VH		Plasma		Mean BP (mmHg)	
Group	Start	End	Start	End	Start	End
A1(n=5)	15(±3)	21(±6)	28(±3)	51(± 8)*	75(± 7)	72(± 4)
A2(n=6)	14(±2)	38(±9)*♦	30(±5)	64(±19)*	63(±18)	37(±16)*♦
B1(n=7)	15(±2)	22(±5)*	25(±5)	83(±28)*	77(±10)	26(±19)*
B2(n=6)	13(±2)	22(±5)*	25(±4)	70(±29)*	92(±19)	30(±22)*

** p<0.05, end vs. start. ♦ p<0.01, A2 vs. A1*

Conclusions: 1) VH Hx rises higher and more rapidly in combined hypoxemia/cerebral ischemia, by contrast with plasma levels. Thus, VH Hx may be derived from both plasma and brain. 2) Biochemical changes are identical in continuous and repetitive models of mild hypoxemia.

▲ 225

GLYCEROL CONTRIBUTES TO GLUCOSE PRODUCTION DURING THE FIRST POSTNATAL DAY IN VERY IMMATURE NEWBORNS (<28W).

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Immature infants have small, rapidly depleted glycogen stores. Therefore, other energy substrates are necessary to meet the metabolic demands. The aim of the present study was to determine production rates of glucose and glycerol, and the degree of conversion of glycerol to glucose in infants born after <28 w.

Subjects and methods: Five infants with birth weights of 665-1173 g and gestational ages of 25-27 w were studied at postnatal ages of 13-24 h. Tracer dilution technique with 6,6-$^2H_2$-glucose and 2-^{13}C-glycerol was used. Production rates of glucose and glycerol were calculated from isotopic enrichments, determined by use of gas chromatography/mass spectrometry. Conversion of glycerol to glucose was calculated from the ^{13}C-enrichment of glucose. Insulin and glucagon were measured by RIA.

Results: The production rate of glucose was 16.4 ± 7.2 and that of glycerol 11.3 ± 6.9 $\mu mol \cdot kg^{-1} \cdot min^{-1}$. The fraction of glycerol converted to glucose was 42.1 ± 16.3 %, corresponding to 16.5 ± 13.8 % of the glucose production rate. The plasma concentration of glucose was 2.5 ± 1.0 mM, that of glycerol 346 ± 454 μM and those of insulin and glucagon 6.6 ± 2.2 $\mu U \cdot mL^{-1}$ and 294 ± 57 $pg \cdot mL^{-1}$, respectively. ($\bar{X}\pm SD$)

Conclusion: Very immature infants are capable to produce glucose and glycerol during their first postnatal day. The results also show, that in these immature infants glycerol contributes to glucose production.

● 226

BACTERIAL COLONIZATION AND POLYMORPHONUCLEAR LEUKOCYTES IN GASTRIC ASPIRATE OF CRITICALLY ILL NEONATES IN THE FIRST THREE DAYS OF LIFE.

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Objective: 1. Estimating the gastric bacterial colonization rate and number of leukocytes of gastric aspirate.2. Evaluating the relations: colonization and leukocytes, colonization and gastric pH. **Patients:** 100 unfed newborns admitted to ICU in the first day of life (mean weight 2120g, mean gestational age 34.2 weeks). **Method:** Gastric aspirates were analyzed for pH, bacterial colonization and number of leukocytes at intervals of 24h. Study protocol was approved by Local Ethical Commitee. **Results:** 1. Significantly higher rate of bacterial colonization was observed between the first and the second day of life (p<0,01). Number of leukocytes increased significantly between the first and the third day (p<0,001). 2. Gastric bacterial colonization was correlated with gastric pH (p<<0,001) but didn't correlated with number of leukocytes (p>0.4). **Conclusions:** Our data confirm the general rule ' the lower gastric pH the less number of bacteria in stomach'. It is interesting to apply this rule to ICU newborns, as well as the fact that in the same group gastric acidity was related to severity of illness.

● 227

PLASMA FREE CARNITINE LEVELS IN CHILDREN WITH MALNUTRITION

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In this study plasma free carnitine and albumin levels were measured in children with protein energy malnutrition (PEM). A total of 71 children with malnutrition were studied. The control group consisted of 20 healthy children.

Mean plasma carnitin level was 78.4±1.94 nmol/ml in the control group. Marasmus and kwashiorkor patients displayed lower plasma free carnitine values which were found statistically significant when compared to those of the control ($P < 0.001$). However those values were significantly lower in kwashiorkor patients than those in marasmic cases ($29.7 > 5.46$). These was no correlation between serum albumin and free carnitine levels in cases with kwashiorkor.

The reason why no correlation between serum albumin and free carnitine levels was observed in the cases investigated in this work has been attributed to varying disfunction of the control mechanism in cases with kwashiorkor.

We concluded that further investigations of carnitine concentration at tissue level are essential in PEM for a more effective manegement of carnitine deficiency by oral means.

▲ 228

POSTASPHYXIAL GLUCOSE ADMINISTRATION AGGRAVATES HYPERINSULINISM IN PIGLETS WITH BILATERAL PNEUMOTHORAX

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Insulin plays a role as a growth factor in the neuronal maturation and it may also have an influence on hypoxic-ischemic brain injuries in newborns. Clinical studies indicated, that hyperinsulinemia with hypoglycemia developed in asphyxiated neonates. Recently, we have also found a severe cerebrospinal fluid (CSF) hyperinsulinism during neonatal asphyxia in piglets. In the present study, we investigated the effect of postasphyxial glucose administration on the insulin levels measured by RIA in the plasma and CSF in newborn pigs. Bilateral pneumothorax (PTX) was induced in 16 piglets; stages of the disease were baseline (B), critical (C) phase with cardiovascular and metabolic failure (arterial hypotension, bradycardia, hypoxemia, combined acidosis), and a 180-min recovery (R) after cardiopulmonary and metabolic (i.v. infusion of $NaHCO_3$ for 15 min, 0.5 mM/l, 10 ml/kg b.w.) resuscitation. 6 piglets (*group 1*) were also given i.v. glucose infusion (1.1 M/l) in the same final volume (10 ml/kg), while the remaining 10 animals (*group 2*) received no glucose. Insulin level was increased in C phase compared to that in B in plasma (1018 ± 231 pM/l vs. 438 ± 81 pM/l, n=16, p<0.001, all values are means ± SEM), but it was unchanged in CSF (38 ± 5 pM/l vs. 42 ± 7 pM/l, n=16, N.S.). A more severe hyperinsulinemia occurred in *group 1* than in *group 2* during R (3157 ± 738 pM/l vs. 1358 ± 495 pM/l, p<0.001 at 60-min-R; and 2211 ± 442 pM/l vs. 534 ± 245 pM/l, p<0.001, at 180-min-R). CSF hyperinsulinism was also significantly (p<0.001) increased in *group 1* compared to that in *group 2* during R (239 ± 77 pM/l vs. 60 ± 13 pM/l, at 60-min-R; and 249 ± 52 pM/l vs. 164 ± 52 pM/l, at 180-min-R). In conclusion, glucose administration increased the asphyxia-induced hyperinsulinism in plasma and CSF of piglets, which may alter the severity of neonatal hypoxic-ischemic brain injuries.