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AMNIOTIC FLUID CHROMIUM (AFCr) IN INFANTS OF DIABETIC MOTHERS. Edward Conner & George Cassady. Div. of Perinatal Medicine, Univ. of Ala. in B'ham, Ala.

Fetal 'leaching' of maternal Cr has been suggested by scattered observations of elevated Cr levels in the term and unusually low Cr levels in the pre-term infant as well as by diminishing maternal Cr levels with increasing gestation and parity. Available data suggest an inverse relation between Cr levels and glucose tolerance. This study represents an ongoing attempt to develop normative data on Cr in the perinatal period.

AF Cr was measured in a graphite furnace (coeff. of var. 11%; recovery rate 95-105%) in 31 AF from 24 patients.

GROUP	n(WOMEN/AF)	AGE	PARITY	L/S	AFCr (range)
Diabetics	9/9	25.4	1.0	2.4*	2.2**(.4-4.2)
High-Risk	6/9	29.0*	1.8	1.8	4.3 (.1-13.3)
C/Section(C/S)	9/13	22.6	3.0	1.9	4.5 (.7-14.9)

(mean values; *$p<.05$ by t test; **$p<.05$ by χ^2)

AF Cr was $\leq$ 4.2 in 9/9 samples from Class B-D diabetics but was >4.2PPB in 7/13 samples from women undergoing repeat C/S.

AF Cr tended to decline with advancing maternal age in C/S ($r= -.33$) but not in other groups. AF Cr was inversely related to L/S in diabetic ($r= -.52$) and high risk groups ($r= -.43$) but not in C/S group. The small sample size precluded statistical significance of these relations.

These preliminary data show an inverse relation of AF Cr to fetal maturity in diabetic and other high risk pregnancies. AF Cr levels are lower in diabetic than in other pregnancies.

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BLUNTED MUSCLE RESPONSIVENESS TO INSULIN IN THE NEONATAL RAT. R.M. Cowett, M. Czech, J.B. Susa, R. Schwartz, W. Oh. Brown Univ. Program in Med., Depts. of Pediatrics and Biochemistry, Providence, R. I.

Peripheral muscle responsiveness to insulin was studied in neonatal rats in vivo by measuring incorporation of labelled glucose to glycogen in diaphragm. ^{14}C glucose (4.2mM) was injected intraperitoneally (100 µl/10gm) with or without (control) insulin(570 mU/ml) into pups from 17 litters of Sprague Dawley rats by the Rafaelson technique. Diaphragms were excised after a 30-minute incubation at birth, 24, 48, 72, or 168 hrs. of age and in 12 adult rats. In control animals $\leq$72 hrs. of age, there was no significant difference in incorporation of labelled glucose. In insulin treated animals there was always an increased incorporation of labelled glucose in comparison to age-matched controls ($p<.025$). The insulin stimulated percent incorporation of labelled glucose was lower at birth (211%) and at 24 hrs. (235%), but increased with advancing age and approached adult levels by 168 hrs. (1815%). At birth and at 24 hrs. a 5 log insulin dose response curve showed significant incorporation only at 570 mU/ml; by 72 hrs. a significant increase was noted at 5.7 mU/ml insulin. Total muscle glycogen was high at birth (1.22gm%), fell to 50% of the birth value from 24 through 168 hrs., and would not account for the decreased insulin response noted. Preliminary studies of glucose transport using ^{14}C deoxyglucose did not fully explain the reduced muscle response to insulin. Our data suggest a blunted responsiveness of muscle to insulin during the 1st 24 hrs.in rats probably related to attenuated intracellular response.

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HIGHER SERUM CARNITINE LEVELS AND KETOGENESIS IN BREAST FED AS COMPARED TO FORMULA FED INFANTS. Edward Curry and Joseph B. Warshaw, Yale University School of Medicine. Department of Pediatrics, New Haven, CT.

The normal newborn infant undergoes a marked metabolic transition in the first days of life. A major feature of this postnatal metabolic adaptation is development of the capacity to utilize fatty acids and ketone bodies as energy substrates. Because of the essential role of carnitine in fatty acid oxidation and ketogenesis, we have investigated relationships between serum carnitine concentrations and serum levels of ketone bodies in breast fed as compared to formula fed newborns. The mean carnitine concentrations in cord blood of normal full-term newborns was 34 nmoles/ml. At 42 hours of age, breast fed newborns demonstrated higher carnitine levels (56 nmoles/ml) as compared with canitine levels of formula fed infants (32 nmoles/ml). Correspondingly, by 42 hours of age, serum ketone body concentrations (β-hydroxybutyrate plus acetoacetate) were higher in breast fed (5.9 nmoles/ml) as compared with formula fed infants (3.0 nmoles/ml). The mean glucose concentrations in both groups of infants showed no significant differences. Carnitine concentrations of isolated samples of human breast milk ranged between 50-100 nmoles/ml and were similar to that of formula (40-80 nmoles/ml) suggesting that the carnitine in breast milk may be better absorbed than carnitine in commercial formulas. The data further suggests a relationship between serum carnitine concentration and ketogenesis in newborn infants.

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INTRINSIC REGULATION OF GLUCOSE OUTPUT BY ISOLATED NEONATAL PIG LIVER. Dolanski, E.A., Bieber, L.L., Olgaard, M.K. and Helmrath, T.A., College of Human Medicine, Michigan State University, Dept. of Human Development, East Lansing.

The aim of the project was to investigate the ability of neonatal liver to autoregulate glucose under various glucose concentrations. Using a recirculating isolated liver perfusion technique, livers from 3-5 day old fasted piglets were perfused for 3 hours with either 0 or 300mg/dl glucose alone or in association with 10mM lactate or alanine. The perfusion system was continuously monitored for temperature, humidity, pH, pCO_2, pO_2 and adjusted as required to mimic physiologic parameters. Serial samples were assayed for glucose, lactate, alanine and glucose (6-^3H). Net production of glucose was significantly higher with 0mg glucose than with 300mg/dl in the perfusate ($p < 0.05$). Glucose utilization by the liver did not vary with the treatments or substrates used. Lactate concentration was monitored and no difference was found between the two treatments. In both instances lactate rose from 0.45mM at the beginning, quickly plateaued at 1-1.3mM in 15-30 minutes, and remained at that level for the 3 hrs. of perfusion. When labeled lactate or alanine was presented to the liver, it was converted to glucose without a significant increase of net glucose production. Liver glycogen before and after perfusion were extremely low and did not change. These data suggest that the neonatal piglet liver has the ability to autoregulate glucose and that this mechanism can be inhibited by exogenous glucose. (Sup. by NIH Grant HD05821-06).

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PRELIMINARY EVALUATION OF A FOUR CHANNEL ELECTROLYTE ANALYZER (PVA-4) FOR PEDIATRIC INSTITUTIONS. Henry Drott & Shlomo Friedman, University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia, Department of Pediatrics, Philadelphia, Pennsylvania.

A PVA-4 instrument was modified to accommodate serum or plasma vols as low as 80µl & compared to a Na&K Flame Photometer, a Cl Coulometric Titrator & a total CO_2(tCO_2) Analyzer. Controls, specimens(spm) & standards were diluted externally 1:5. The reproducibility was tested by analyses of macrodiluted samples of an aq. standard for Na, K & Cl & of individually microdiluted samples. A mean difference ($\pm$1SD)(mEq/L) between [Na]+[K]&[Cl] was 0.14(1.63) for 15 readings with a $p<.83$ & 1.53(1.27) for 22 readings with a $p<.0001$ respectively. The means ($\pm$1SD) of 33 readings of our quality control(QC) material for Na, K & Cl by the PVA-4 were 128.3(1.29), 3.80(.068) & 99.4(1.14) compared to the QC Program means by equivalent methodologies of 129.01(1.57), 3.69(.09) & 99.84(1.34) for Level I respectively. Level II means for 39 readings were 145.8(1.07), 6.41(.052) & 114.7(.86) compared to 146.65(1.57), 6.39(.13) & 114.4(1.29) respectively. Linear regression analysis of 23 patients(pts) spm for Na, K&Cl resulted in correlation coefficients(r) of 0.883, 0.972 & 0.902 respectively. The PVA-4 HCO_3 data for 22 pt spm showed good correlation to the calculated HCO_3($r=0.981$) & to the tCO_2($r=.932$). However, alkalinity/acidity in pt sera resulted in an over/under estimation of HCO_3 e.g. [HCO_3] of 39 & 5.6 compared to [CO_2] of 33 & 9 respectively. The major drawbacks of the PVA-4 are the misleading HCO_3 values in pt sera with pH imbalance & the additional external dilution of spm.

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EFFECTS OF GLUCAGON (G) AND INSULIN (I) ON SERUM CALCIUM (Ca_s) IN NEWBORN AND ADULT RATS. Haim Elrad, Turkan Dagoglu and William H. Bergstrom. Depts. of Peds. and OB-GYN, SUNY, Upstate Med. Ctr., Syracuse, N.Y.

In adults of several species including man, a small transient decrease in Ca_s ($-\Delta Ca_s$) follows G doses of 1-10 ug/gm. The effects of maturation and I on this phenomenon were studied in newborn (7 gm, Grp. A) and adult (200 gm., Grp. B) rats. In grp. A $-\Delta Ca_s$ was maximal 60' after 1 ug/gm of G. Adding .01 U/gm of I to this dose of G reduced ΔCa_s from -1.75 mg/dl to -0.74 mg/dl. In Grp. B no ΔCa_s was seen 60' after 1 ug/gm of G. After pancreatectomy (PNX) and same dose of G, ΔCa_s was -1.23 mg/dl. Adding I (.01 U/gm) prevented the effect of G on Ca_s.

Group	Treatment	ΔCa_s, mg/dl	n	p
A	G, 1 ug/gm IP	-1.75±.23	11	<.01
A	G, 1 ug/gm IP+I .01 U/gm IM	-0.74±.10	19	<.01
B	G, 1 ug/gm IP	- .07±.14	24	>.1
B	G, .5 ug/gm IV	+ .19±.18	10	>.1
B	PNX+G 0.5 ug/gm IV	-1.23±.10	10	<.01
B	PNX+G 0.5 ug/gm IV+I .01 U/gm	-0.14±.11	6	>.1

Insulin apparently accounts for most of the decrease in post-glucagon ΔCa_s with maturation. The mechanism by which G affects Ca_s is not known, and the physiological and clinical significance of this phenomenon are presently obscure.