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RELATIONSHIP BETWEEN DEXTROSTIX, PLASMA AND WHOLE BLOOD GLUCOSE IN THE RANGE 0 - 100 MG/DL, Virginia D. Black, Keith B. Hammond and Craig P. Black (Spon. by Lula O. Lubchenco). University of Colorado Medical Center, Division of Perinatal Medicine, Denver.

Two laboratory measurements frequently made in the newborn period are Dextrostix (Ames) and hematocrit; yet the relationship between Dextrostix, plasma glucose and whole blood glucose values has not been described. Umbilical vein and peripheral vein blood samples were studied for whole blood, plasma and red cell glucose (YSI Model 23A) and were compared to values obtained using the Dextrostix read by eye. Dextrostix predicted whole blood glucose (R = .83, slope of regression = 1.1, not significantly different from 1.0). Plasma glucose was underestimated by the Dextrostix (R = .64, slope = .70 in the range measured in vivo (30-100 mg.dl^{-1}). Measurements in vitro were carried out using umbilical vein blood during incubation with and without added glucose to determine the relationship between plasma and whole blood glucose at the lower glucose levels. No statistically significant difference was observed between plasma and whole blood glucose at these low levels. Dextrostix reflected both of these levels. No clinically significant error would have been made using the Dextrostix.

In vivo, red cell glucose fell more quickly than plasma glucose at values greater than 100. Below that level, plasma and red cell glucose fell at similar rates. However, preliminary data from infants with polycythemia/hyperviscosity reveal red cell glucose values lower than would be predicted by the plasma glucose value alone.

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CALCITONIN THERAPY IN OSTEOGENESIS IMPERFECTA, S. Castells, C. Chakrabarti, R.S. Bachtell, C. Colbert, and S. Yasumura, Dept. of Peds., and Physiol. SUNY, Downstate Med. Ctr., B'klyn., N.Y. and Radiological Res. Lab., Greene Memorial Hosp., Yellow Springs, Ohio

We have previously shown increased skeletal turnover in O.I. with resorption being more accelerated than bone accretion. Calcitonin, through its ability to inhibit skeletal resorption, might be useful in the treatment of O.I. Twenty-five patients with O.I. tarda and 25 with O.I. congenita received synthetic salmon calcitonin (SSC) 2 MRC units/kg I.M. three times a week, for periods of 6 to 48 months. In O.I.T. the fracture rate decreased from 2.7 before calcitonin to 0.5/yr/patient during calcitonin; in O.I.C. from 3.6 to 0.8/yr/patient. Therapy was discontinued in 3 patients because of persistent vomiting and in one because of an allergic skin reaction. X-rays of the left hand with the same control metal wedge were taken every 3 months. Bone density was measured by a photodensitometer connected to a LINC-8 computer, and corrected for changes between X-rays and compared to normals for age and sex. 38/50 studies were successfully terminated. Calcitonin significantly increased bone density in the 0-5 year age range when compared with normal rates. SSC appears to be more effective in increasing bone density during the first year of therapy. Antibodies to SSC were found in 6 patients treated with SSC, at titers of 1:10 to 1:600. There was no increase in plasma iPTH concentrations during SSC administration. These data suggest that long term SSC therapy reduces fracture-rate and increases bone density in young children with O.I. (Supported by NIH Grant RR-318)

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FATTY ACID (FA) AND PROSTAGLANDIN (PG) LEVELS IN CHILDREN WITH DIABETES MELLITUS. H. Peter Chase, R. L. Williams and Jacqueline Dupont. Univ. of Colo. Med. Ctr., Dept. of Ped., Denver and Colo. State Univ., Dept. of Food Sci. and Nutr., Fort Collins, Colo.

Fatty acid levels and PG production were measured in blood from 40 test children with diabetes mellitus and 20 control children. Test children ranged from 3 to 19 years of age, had required insulin for at least 1 year and were considered well when tested as outpatients. Blood was drawn from all participants following a 10 to 12 hr overnight fast.

The percent plasma FA's varied in that stearic acid (18:0) levels were higher and oleic acid (18:1Δ9) levels lower in the diabetic compared to the control children. Quantitative levels (mg/dl) of most FA's were elevated in the plasma of the diabetic children with total FA levels of 200 ± 45 mg/dl compared to 174 ± 27 mg/dl for the control children ($p<.01$). Red blood cell FA's and plasma and RBC phospholipids were similar in the 2 groups.

Prostaglandin E_2 and $F_{2\alpha}$ levels (measured by radioimmunoassay) were higher in the diabetic than in the control children following 10,20 or 70 minutes incubation of whole blood at 37°C, whereas PGE_1 levels were higher only after 20 minutes incubation. Prostaglandin values did not correlate with 24 hr urine sugars or fasting blood sugars. The elevated PG levels may be related to the increased blood coagulation and/or the kidney or eye complications associated with diabetes mellitus.

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SERUM 1,25-$(OH)_2$-VITAMIN D_3 LEVELS IN CHILDREN AND ALTERATION WITH DISORDERS OF VITAMIN D METABOLISM Russell W. Chesney, Alan Hamstra and Hector F. DeLuca The Department of Pediatrics and Biochemistry, University Hospitals, The University of Wisconsin, Madison, Wisconsin

Using the Eisman competitive binding assay, the endogenous serum concentrations of the most active vitamin D metabolite, 1,25-$(OH)_2D_3$ was measured in healthy children and in patients with 3 conditions known to impair vitamin D metabolism. The mean value in 56 children aged 2 to 15 years was 43.6 ± 3 (S.E.) pgm/ml; the level in 30 boys was 42.9 ± 6.3 and in 26 girls 44.3 ± 4.2 pgm/ml. These values are higher than the values reported in term newborns by Steichen et al. (J. Pediatr., in press) of 21 ± 2 pgm/ml. (n=28) and in adults aged 50-80 years reported by Eiseman (29 ± 2 pgm/ml) ($p<.01$). The concentration in children with uremic osteodystrophy while on D_2 therapy was 15 ± 6 pgm/ml and rose to 53 ± 13 ($p<.05$) after treatment with 1,25-$(OH)_2D_3$ at 14-36 pgm/kg/day. Simultaneous serum calcium values rose from 7.5 ± 1.6 (S.D.) mg/dl pretherapy to 9.9 ± 0.4 mg/dl. Values in 2 patients with the Fanconi syndrome were 0 and 2 pgm/ml and rose to 34 ± 5 (S.E.) with improvement in rickets. 2 patients with hypoparathyroidism had values of 16 and 18 pgm/ml which rose to 76 ± 10 pgm/ml after 1 ugm 1,25 $(OH)_2D_3$ daily. These results support the notion that an age related increase in 1,25-$(OH)_2D_3$ is present and that these levels reach a peak during childhood. In addition, in 3 conditions associated with impaired vitamin D metabolism circulating levels are reduced, but rise with provision of oral 1,25 $(OH)_2D_3$ therapy.

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INTEGUMENTARY LOSS OF CALCIUM. Jen-Yih Chu, Sheldon Margen, Doris Calloway, and Francoise Costa. (Spon. by James A. Monteleone). St. Louis University School of Medicine, Department of Pediatrics, St. Louis, MO and University of California, Department of Nutritional Sciences, Berkeley, CA.

The amount of integumentary calcium loss has been generally considered very small and therefore, has rarely been included in calcium metabolic studies. During our study of calcium and protein metabolism, two conditions of integumentary calcium loss were measured. The first, collected over a six-day period represented dermal loss by an ambulatory but rather sedentary man. The second was collected over a 40 minute period of strenous exercise. The daily loss of 16 subjects in 52 determination of six-day periods each was 8.7 ± 1.9 mg/m^2/day. The amount was not influenced by calcium intake (0.1 to 2.3 g/day). In contrast to urinary calcium excretion, which is directly related to protein intake, there was no significant change by varing protein intake (1 to 96 g nitrogen/day). No compensatory relationship between urinary and integumentary calcium excretion was noted. During strenous exercise calcium loss increased to to 25 mg (11 to 45 mg) in 40 minutes. There was no compensatory decrease in urinary excretion on the day of strenous exercise. It was also noted that this calcium loss was not affected by general calcium balance. The amount of integumentary loss may become even greater than the urinary calcium excretion. We conclude that integumentary calcium loss probably should be considered in calcium metabolic studies.

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URINARY H^+EXCRETION IN VERY LOW BIRTHWEIGHT (VLBW) INFANTS. EFFECT OF ALTERING Ca AND P INTAKE. Raul F. Cifuentes, J. Williamson Balfe, Ingeborg C. Radde, Graham W. Chance. Dept. Paediatrics, Hosp. for Sick Children, Toronto, Canada.

To determine the response of VLBW infants (<1.3 kg) to changes in Ca and P intake (postnatal age 2 to 6 wks), we measured net acid excretion (NAE), titratable acidity (TA) and NH_4^+ on 24-hr urines collected on 45 thriving VLBW infants receiving SMA S26 (Wyeth Corp.), 80 cal/dl, 200 ml/kg/24 hr daily. The Ca/P intake (mg/kg) was: Group 1: 100/80; Gp. 2: 250/80; Gp. 3: 210/80; Gp. 4: 210/120. Gps. 1 and 2 received a Na intake of 1.5 mEq/kg/24 hr; Gps. 3 and 4 3 mEq/kg/24 hr, the difference as bicarbonate. Table shows results (mean±SEM) (*$p<0.001$) for baseline (2-3 wks) and study collections (4-6 wks).

Age	μEq/min/m^2	Gp. 1 (n=9)	Gp. 2 (9)	Gp. 3 (14)	Gp. 4 (13)
2-3 wks	TA	6.9±0.5	6.7±0.9	1.2±1.0	2.5±1.7
	NH_4^+	10.1±1.2	10.9±1.4	5.9±0.6	6.4±0.6
	NAE	17.0±1.2	17.6±1.9	7.1±1.3	8.9±1.8
4-6 wks	TA	7.9±0.9	5.0±0.7	0.7±1.1	10.4±1.2*
	NH_4^+	12.6±1.1	10.1±1.1	6.0±0.5	8.9±0.7
	NAE	19.6±1.9	15.1±1.6	6.7±1.0	19.3±1.5*

Despite the 4-fold increase in Ca net retention in Gp. 2 compared to Gp. 1, H^+excretion did not increase. Added $NaHCO_3$ decreased TA and NH_4^+, and P-supplements increased TA and NAE. TA and urinary P-excretion were correlated in Gps. 3 and 4 ($y=-0.24+0.33x$)(*). Thus, VLBW infants respond appropriately to $NaHCO_3$ and P-supplement with respect to H-ion excretion.